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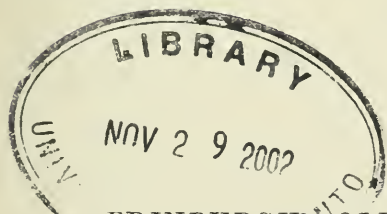
THE TRANSACTIONS  
OF THE  
MEDICO-CHIRURGICAL SOCIETY OF  
EDINBURGH.

VOL. XV.—NEW SERIES.

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SESSION 1895-96.

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EDINBURGH: OLIVER AND BOYD,  
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## ERRATUM.

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The two Plates facing page 156 should be placed opposite page 256 to illustrate Dr Ritchie's Paper on OSTEOMALACIA CARCINOMATOSA.





## P R E F A C E.

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THE present Volume is the *Fifteenth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers ; the exhibition of Patients, illustrating rare and interesting forms of disease ; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

In an Appendix will be found papers by Professor John Struthers "On Separate Acromion Process simulating Fracture," and by Professor T. R. Fraser and Dr Alexander Bruce "On a Case of Diabetic Neuritis."

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

WILLIAM CRAIG,  
*Editor.*

October 1896.



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	Alexander John Keiller, L.R.C.P. & S. Ed., <i>North Berwick,</i>	1889
335	D. C. Braidwood, M.B., C.M., <i>Halkirk, Caithness,</i>	1889
	Professor J. Berry Haycraft, M.D., D.Sc., <i>Cardiff,</i>	1889
	Professor A. W. Hughes, M.B., F.R.C.S. Ed., M.R.C.S. Eng., <i>Cardiff,</i>	1889
	Hugh Jamieson, M.B., C.M.,	1889
	Albert E. Morison, M.B., F.R.C.S. Ed., M.R.C.S. Eng., <i>Hartlepool,</i>	1889
340	William H. Barrett, M.B., C.M., <i>Southport,</i>	1890
	James Hunter, M.D., C.M., <i>Linlithgow,</i>	1890
	George M. Robertson, M.B., F.R.C.P. Ed., <i>Murthly,</i>	1890
	Charles Templeman, M.D. C.M., <i>Dundee,</i>	1891
	John Macpherson, M.D., F.R.C.P. Ed., <i>Larbert,</i>	1891
345	J. J. Douglas, M.D., F.R.C.P. Ed., <i>London,</i>	1891
	Robert Stirling, M.B., C.M., <i>Perth,</i>	1891
	Simson C. Fowler, M.B., C.M., <i>Juniper Green,</i>	1892
	Robert Dundas Helm, M.D., C.M., <i>Carlisle,</i>	1892
	William Gordon Woodrow Sanders, M.B., F.R.C.P. Ed., <i>Cuen,</i>	1892
350	W. Ramsay Smith, M.B., C.M., <i>South Australia,</i>	1892
	T. Herbert Littlejohn, M.B., F.R.C.S. Ed., <i>Scarborough,</i>	1892
	Alexander Peyer, M.D., <i>Zürich,</i>	1893
	Alexander Reid Urquhart, M.D., F.R.C.P. Ed., <i>Perth,</i>	1893
	F. W. Foxcroft, M.B., C.M., <i>Wilmslow,</i>	1893
355	William B. Mackay, M.D., M.R.C.S. Eng., <i>Berwick-on-Tweed,</i>	1893
	Alex. Mitchell Stalker, M.D., C.M., <i>Dundee,</i>	1893
	D. W. Johnston, F.R.C.S. Ed., <i>Johannesburg, S. Africa,</i>	1893
	William Brendon T. Gubbin, M.D., C.M., <i>Bardolph, Hertford,</i>	1893
	Frank Ashby Elkins, M.D., C.M., <i>Sunderland,</i>	1893
360	Philip Grierson Borrowman, M.B., C.M., <i>Elie,</i>	1893
	William Craig, M.B., C.M., <i>Cowdenbeath,</i>	1894
	James Mackenzie, M.D., C.M., <i>Burnley,</i>	1894
	Charles E. Douglas, M.D., C.M., <i>Cupar-Fife,</i>	1894
	Thomas Easton, M.D. C.M., <i>Stranraer,</i>	1894
365	William Simmers, M.B., C.M., <i>Crail,</i>	1894
	Gopal Govind Vathe, M.D., <i>Bombay,</i>	1895
	John Hosack Fraser, M.B., M.R.C.P. Ed., <i>Bridge of Allan,</i>	1895

		Date of Admission.
	John Struthers, M.B., C.M., <i>South Africa</i> , . . . . .	1895
	George Thomas Beatson, M.D., C.M., <i>Glasgow</i> , . . . . .	1895
370	Andrew Balfour, M.B., C.M., . . . . .	1895
	Robert Durward Clarkson, M.B., C.M., <i>Falkirk</i> , . . . . .	1896

## ORDINARY MEMBERS.

## ARRANGED ALPHABETICALLY.

*(a.)* Members who pay the Annual Subscription of  
Ten Shillings.

	Dr R. Abernethy, 10 St Colme Street, . . . . .	1892
	Dr J. O. Affleck, 38 Heriot Row, . . . . .	1871
	Dr R. S. Aitchison, 74 Great King Street, . . . . .	1887
	Dr D. Aitken, 152 Packington Street, Islington, London, N., . . . . .	1887
5	Dr W. A. J. Alexander, 13 Gayfield Square, . . . . .	1896
	Dr James Andrew, 2 Atholl Crescent, . . . . .	1869
	Professor Annandale, 34 Charlotte Square, . . . . .	1863
	Dr E. F. Armour, 149 Bruntsfield Place, . . . . .	1890
	Dr G. W. Balfour, 17 Walker Street, . . . . .	1874
10	Dr J. W. Ballantyne, 24 Melville Street, . . . . .	1885
	Dr A. H. Freeland Barbour, 4 Charlotte Square, . . . . .	1881
	Joseph Bell, Esq., 2 Melville Crescent, . . . . .	1862
	Dr G. A. Berry, 31 Drumshengh Gardens, . . . . .	1883
	Dr R. J. A. Berry, 4 Howard Place, . . . . .	1893
15	Dr Alexander Black, 13 Howe Street, . . . . .	1883
	Dr W. T. Black, 2 George Square, . . . . .	1877
	Dr Robert H. Blaikie, 42 Minto Street, . . . . .	1883
	Dr Bleloch, 2 Lonsdale Terrace, . . . . .	1871
	Dr G. P. Boddie, 147 Bruntsfield Place, . . . . .	1888
20	William Booth, Esq., 1 Minto Street, . . . . .	1888
	Dr F. D. Boyd, 6 Atholl Place, . . . . .	1891
	Dr Byrom Bramwell, 23 Drumsheugh Gardens, . . . . .	1876
	Dr N. T. Brewis, 23 Rutland Street, . . . . .	1886
	Dr J. Graham Brown, 3 Chester Street, <i>Secretary</i> , . . . . .	1878
25	Dr J. Murdoch Brown, 9 Walker Street, . . . . .	1885
	Dr Alexander Bruce, 13 Alva Street, . . . . .	1883
	Dr Lewis C. Bruce, Royal Asylum, Morningside, . . . . .	1895
	Dr Buist, 1 Clifton Terrace, . . . . .	1877
	Dr T. M. Burn-Murdoch, 14 Charlotte Square, . . . . .	1886
30	Dr Cadell, 22 Ainslie Place, . . . . .	1870
	Dr Francis M. Caird, 21 Rutland Street, . . . . .	1883
	Dr H. L. Calder, 60 Leith Walk, . . . . .	1884
	Dr James Cameron, 13 Fettes Row, . . . . .	1895
	Dr Cappie, 37 Lauriston Place, . . . . .	1855
35	Dr Edward Carmichael, 21 Abercromby Place, . . . . .	1887
	Dr J. Carmichael, 22 Northumberland Street, <i>Vice-President</i> , . . . . .	1870
	Dr C. W. Cathcart, 8 Randolph Crescent, . . . . .	1883
	Dr J. G. Cattanach, 3 Alvanley Terrace, . . . . .	1895
	Dr T. F. S. Caverhill, 16 Randolph Crescent, . . . . .	1883
40	Professor John Chiene, 26 Charlotte Square, . . . . .	1867
	Dr J. A. Clark, 4 Cambridge Street, . . . . .	1893
	Dr Church, 36 George Square, . . . . .	1876
	Dr Clouston, Tipperlinn House, Morningside Place, . . . . .	1861
	Dr Cotterill, 24 Manor Place, . . . . .	1878

		Date of Admission.
45	Dr William Craig, 71 Bruntsfield Place, <i>Vice-President</i> , . . . . .	1869
	Dr Halliday Croom, 25 Charlotte Square, . . . . .	1870
	Dr G. Matheson Cullen, 48 Lauriston Place, . . . . .	1892
	Dr A. S. Cumming, 18 Ainslie Place, . . . . .	1884
	Dr John Cumming, 94 Gilmore Place, . . . . .	1894
50	Dr R. J. B. Cunynghame, 18 Rothesay Place, . . . . .	1868
	Dr T. B. Darling, 13 Merchiston Place, . . . . .	1887
	Dr M. Dewar, 24 Lauriston Place, . . . . .	1885
	Dr George Dickson, 9 India Street, . . . . .	1884
	Dr Kenneth M. Douglas, 26 Rutland Street, . . . . .	1888
55	Dr J. W. Dowden, 22 Melville Street, . . . . .	1893
	Dr John Duncan, 8 Ainslie Place, . . . . .	1868
	Dr Kirk Duncanson, 22 Drumsheugh Gardens, . . . . .	1871
	Dr John A. H. Duncan, 32 Morningside Drive, . . . . .	1895
	Dr H. M. Dunlop, 20 Abercromby Place, . . . . .	1883
60	Dr J. C. Dunlop, 24 Stafford Street, . . . . .	1892
	Dr J. Dunsmore, 53 Queen Street, . . . . .	1872
	Dr William Elder, 4 John's Place, Leith, . . . . .	1892
	Dr R. W. Felkin, 8 Alva Street, . . . . .	1885
	Dr J. Haig Ferguson, 25 Rutland Street, . . . . .	1885
65	Dr W. A. Finlay, St Helen's, Russell Place, Trinity, . . . . .	1875
	Dr Andrew Fleming, 8 Napier Road, . . . . .	1880
	Dr R. A. Fleming, 36 Drumsheugh Gardens, . . . . .	1890
	Dr Foulis, 34 Heriot Row, . . . . .	1875
	Dr J. S. Fowler, 42 Henderson Row, . . . . .	1895
70	Dr John Fraser, 19 Strathearn Road, . . . . .	1878
	Professor Thomas R. Fraser, 13 Drumsheugh Gardens, . . . . .	1865
	Dr Garland, 53 Charlotte Street, Leith, . . . . .	1873
	Dr G. A. Gibson, 17 Alva Street, . . . . .	1880
	Dr A. B. Giles, 1 Kew Terrace, . . . . .	1893
75	Dr A. Lockhart Gillespie, 23 Walker Street, . . . . .	1891
	Dr F. M. Graham, Cowgate Dispensary, . . . . .	1894
	Dr J. Allan Gray, 107 Ferry Road, . . . . .	1879
	Professor Greenfield, 7 Heriot Row, . . . . .	1886
	Dr David Greig, 38 Coates Gardens, . . . . .	1854
80	Dr David M. Greig, 25 Tay Street, Dundee, . . . . .	1892
	Dr G. L. Gulland, 6 Alva Street, . . . . .	1888
	Dr A. C. Guthrie, 171 Constitution Street, Leith, . . . . .	1890
	Dr William Guy, 11 Wemyss Place, . . . . .	1890
	Dr William Haldane, Viewforth, Bridge of Allan, . . . . .	1889
85	Dr John Hardie, 12 Newington Road, . . . . .	1895
	Dr D. Berry Hart, 29 Charlotte Square, . . . . .	1886
	Dr James Harvey, 7 Blenheim Place, . . . . .	1893
	Dr Henry Hay, 7 Brandon Street, . . . . .	1884
	Dr John Henderson, 7 John's Place, Leith, . . . . .	1848
90	Mr J. W. B. Hodsdon, 52 Melville Street, <i>Secretary</i> , . . . . .	1883
	Dr George Hunter, 33 Palmerston Place, . . . . .	1876
	Dr Husband, 4 Royal Circus, . . . . .	1849
	Dr J. Hutcheson, 44 Moray Place, . . . . .	1890
	Dr Robert Inch, Gorebridge, . . . . .	1887
95	Dr R. W. Inkster, 38 Montgomery Street, . . . . .	1895
	Dr W. Wotherspoon Ireland, Mavisbush House, Polton, . . . . .	1893
	Dr Alex. James, 10 Melville Crescent, . . . . .	1877
	Dr Allan Jamieson, 35 Charlotte Square, . . . . .	1876
	Dr James Jamieson, 43 George Square, . . . . .	1877
100	Dr G. M. Johnston, 7 Wellington Place, Leith, . . . . .	1888
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens, <i>Treasurer</i> , . . . . .	1883
	Dr C. Kennedy, 43 Minto Street, . . . . .	1886
	Dr C. B. Ker, 4 Howard Place, . . . . .	1894
	Dr George Kerr, 6 St Colme Street, . . . . .	1885
105	Dr J. H. A. Laing, 11 Melville Street, . . . . .	1889
	Dr Thomas Lawson, 16 Dean Terrace, . . . . .	1893

	Date of Admission.
	1886
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160	1877
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165	1895
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	1859

		Date of Admission.
	Dr A. T. Sloan, 22 Forth Street, . . . . .	1885
170	Dr Andrew Smart, 35 Lauriston Place, . . . . .	1865
	Dr G. D. Smith, 148 Ferry Road, . . . . .	1877
	Dr James Smith, 1 Parson's Green Terrace, . . . . .	1891
	Dr John Smith, 11 Wemyss Place, . . . . .	1856
	Dr J. Cormack Smith, 9 Brunton Place, . . . . .	1896
175	Dr William Smith, 14 Hartington Gardens, . . . . .	1890
	Dr John Stevens, 3 Shandon Crescent, . . . . .	1892
	Professor Sir T. Grainger Stewart, 19 Charlotte Square, . . . . .	1861
	Dr J. S. Stewart, 15 Merchiston Place, . . . . .	1885
	Dr Robert Stewart, 42 George Square, . . . . .	1892
180	Dr William Stewart, 146 Ferry Road, Leith, . . . . .	1889
	Dr H. J. Stiles, 5 Castle Terrace, . . . . .	1889
	Dr S. Stirling, 4 Coates Crescent, . . . . .	1891
	Dr R. Stockman, 12 Hope Street, . . . . .	1891
	Dr A. Stodart-Walker, 30 Walker Street, . . . . .	1894
185	Dr C. A. Sturrock, Dunfermline, . . . . .	1894
	Dr Allan C. Sym, 144 Morningside Road, . . . . .	1889
	Dr William G. Sym, 50 Queen Street, . . . . .	1889
	Dr W. Taylor, 12 Melville Street, . . . . .	1871
	Dr C. C. Teacher, 16 Newington Road, . . . . .	1887
190	Dr C. H. Thatcher, 8 Melville Crescent, . . . . .	1876
	Dr R. Thin, 38 Albany Street, . . . . .	1890
	Dr Alexis Thomson, 32 Rutland Square, . . . . .	1887
	Dr John Thomson, 14 Coates Crescent, . . . . .	1887
	Dr John Tod, 93 Ferry Road, Leith, . . . . .	1895
195	Dr Batty Tuke, 20 Charlotte Square, . . . . .	1864
	Dr Dawson Turner, 37 George Square, . . . . .	1890
	Dr Logan Turner, 2 Coates Crescent, . . . . .	1892
	Dr Underhill, 8 Coates Crescent, . . . . .	1872
	Dr Norman Walker, 7 Manor Place, . . . . .	1891
200	Dr D. Wallace, 66 Northumberland Street, . . . . .	1887
	Dr Douglas Watson, 19 Rutland Street, . . . . .	1894
	Dr P. H. Watson, 16 Charlotte Square, . . . . .	1856
	Dr A. D. Webster, Belleville Lodge, S. Blacket Place, . . . . .	1883
	Dr George R. Wilson, Mavisbank, Polton, . . . . .	1892
205	Dr J. Lockhart Wilson, Duns, . . . . .	1888
	J. L. Wilson, Esq., 4 Buccleuch Place, . . . . .	1883
	Dr T. D. Wilson, 10 Newington Road, . . . . .	1880
	Dr Russell E. Wood, 9 Darnaway Street, . . . . .	1883
	Dr W. Fraser Wright, Bonnington Mount, Bonnington Ter., . . . . .	1894
210	Dr Hamilton Wylie, 1 George Place, . . . . .	1883
	Dr John Wylie, 44 Charlotte Square, <i>Vice-President</i> , . . . . .	1868
	Dr J. Y. Simpson Young, 8 Alva Street, . . . . .	1891
	Dr P. A. Young, 25 Manor Place, . . . . .	1870
	Dr R. J. Erskine Young, 8 Alva Street, . . . . .	1892

(b.) **Members Exempted under Rule V. from paying the Annual Subscription.**

215	Dr D. H. Anderson, 14 <i>Hartington Street, Barrow-in-Furness</i> , . . . . .	1887
	Dr Archibald, 2 <i>The Avenue, Beckenham, Kent</i> , . . . . .	1882
	Dr J. A. Armitage, 28 <i>Waterloo Road South, Wolverhampton</i> , . . . . .	1887
	Dr W. Badger, <i>Penicuik</i> , . . . . .	1882
	Dr J. J. Bailey, <i>Piccadilly Club, London, W.</i> , . . . . .	1874
220	Dr Edwin Baily, <i>Oban</i> , . . . . .	1883
	Dr Andrew Balfour, <i>Portobello</i> , . . . . .	1874
	Dr Andrew Balfour, . . . . .	1895



		Date of Admission.
	Dr James Craig Balfour, 17 Walker Street, . . . . .	1884
	Dr J. H. Balfour, Portobello, . . . . .	1881
225	Dr Alexander Ballantyne, Dalkeith, . . . . .	1872
	Dr W. H. Barrett, 29 Park Crescent, Hesketh Park, Southport,	1890
	Dr F. W. Barry, Local Government Board, Whitehall, London, W., . . . . .	1878
	Dr George T. Beatson, 7 Woodside Crescent, Glasgow, . . . . .	1895
	Surgeon-Captain C. H. Bedford, Bengal Army, care of W. Watson & Co., 28 Apollo Street, Bombay, . . . . .	1889
230	Dr Benjamin D. C. Bell, Kirkwall, . . . . .	1889
	Dr G. J. H. Bell, Bengal Army, . . . . .	1884
	Dr G. H. Bentley, Kirkliston, . . . . .	1877
	Dr J. S. Beveridge, Lochinver, . . . . .	1861
	Dr J. W. Black, 15 Clarges Street, Piccadilly, London, W.,	1865
235	Dr P. G. Borrowman, Elie, Fife, . . . . .	1893
	Dr D. C. Braidwood, Halkirk, Caithness, . . . . .	1889
	Dr David Brodie, 12 Patten Road, Wandsworth Common, London, S.W., . . . . .	1865
	Dr Andrew Brown, 1 Bartholomew Road, Kentish Town, London, N.W., . . . . .	1884
	Dr John Brown, 68 Bank Parade, Burnley, Lancashire, . . . . .	1878
240	Dr J. Macdonald Brown, 48 Mildmay Park, London, N., . . . . .	1883
	Dr W. Watson Campbell, Duns, . . . . .	1887
	Dr R. D. Clarkson, Falkirk, . . . . .	1896
	Professor Cleland, The University, Glasgow, . . . . .	1864
	Dr Coghill, Ventnor, Isle of Wight, . . . . .	1870
245	Dr A. R. Coldstream, 24 Lung Arvo Navvo, Florence, Italy,	1878
	Dr John Connel, Peebles, . . . . .	1876
	Dr William Craig, Cowdenbeath, Fife, . . . . .	1894
	Dr J. R. Crease, 2 Ogle Terrace, South Shields, . . . . .	1885
	Dr P. M. Deas, Wonford House, Exeter, . . . . .	1888
250	Dr Archibald Dickson, Hartree House, Biggar, . . . . .	1871
	Dr A. Halliday Douglas, . . . . .	1842
	Dr A. Home Douglas, Rue Blacas, Nice, France, . . . . .	1889
	Dr C. E. Douglas, Cupar-Fife, . . . . .	1894
	Dr J. J. Douglas, 15 Church Road, Upper Norwood, London,	1891
255	* Dr W. B. Dow, Dunfermline, . . . . .	1879
	Dr Thomas Easton, Hanover House, Stranraer, . . . . .	1894
	Dr J. W. Eastwood, Dinsdale Park, Darlington, . . . . .	1871
	Dr F. A. Elkins, The Asylum, Sunderland, . . . . .	1893
	Sir R. B. Finlay, Q.C., M.P., Middle Temple, London, . . . . .	1864
260	Dr Simon C. Fowler, Juniper Green, . . . . .	1892
	Dr F. W. Foxcroft, Wilmslow, Cheshire, . . . . .	1893
	Dr Dyce Fraser, Chili, . . . . .	1883
	Dr J. Hosack Fraser, Bellfield, Bridge of Allan, . . . . .	1895
	Dr R. Freeland, Broxburn, . . . . .	1879
265	Professor Gamgee, 8 Avenue de la Garce, Lausanne, Switzerland, . . . . .	1863
	Dr William Gayton, Bartram Lodge, Fleet Road, Hampstead, London, N.W., . . . . .	1886
	Dr W. C. Greig, Tangier, Morocco, . . . . .	1884
	Dr Groesbeck, Cincinnati, . . . . .	1875
	Dr W. B. T. Gubbin, Bardolph, near Hertford, . . . . .	1893
270	His Excellency Dr R. H. Gunning, 12 Addison Crescent, Kensington, London, W., . . . . .	1846
	Dr John Haddon, Honeyburn, Hawick, . . . . .	1883
	Dr Archibald Hall, Montreal, . . . . .	1853
	Professor D. J. Hamilton, The University, Aberdeen, . . . . .	1876
	Dr J. W. Hamp, Penn Road, Wolverhampton, . . . . .	1887
275	Dr A. W. Hare, Keyworth, Nottingham, . . . . .	1883
	Dr J. Home-Hay, Alloa, . . . . .	1880
	Professor J. Berry Haycraft, 1 St Andrew's Place, Cardiff, . . . . .	1889

		Date of Admission.
	Dr Stanley Haynes, <i>Malvern, Worcestershire,</i>	1864
	Dr R. Dundas Helm, 3 <i>Alfred Street N., Portland Square,</i>	
	<i>Carlisle,</i>	1892
280	Dr R. E. Horsley, <i>Jud-Falls, Stoncyhurst, Lancashire,</i>	1886
	Dr J. S. Howden, <i>Montrose,</i>	1856
	Professor A. W. Hughes, <i>Cardiff,</i>	1889
	Dr James Hunter, <i>St Catherine's, Linlithgow,</i>	1890
	Dr W. Hunter, 54 <i>Harley Street, Cavendish Square,</i>	
	<i>London, W.,</i>	1887
285	Dr J. Carlyle Johnstone, <i>The Asylum, Melrose,</i>	1882
	Dr Hugh Jamieson, 13 <i>Lauriston Place,</i>	1889
	Dr D. W. Johnston, <i>P.O. Box 2022, Johannesburg, South</i>	
	<i>Africa,</i>	1893
	Dr James Johnston, 53 <i>Princes Square, Bayswater, London,</i>	
	<i>W.,</i>	1871
	Dr J. Keay, <i>District Asylum, Inverness,</i>	1887
290	Dr A. J. Keiller, <i>North Berwick,</i>	1889
	Dr George Keith, <i>Moidart Cottage, Currie,</i>	1845
	Dr Skene Keith, 42 <i>Charles Street, Berkeley Square,</i>	
	<i>London, W.,</i>	1885
	Dr W. Scott Lang,	1886
	Dr Harvey Littlejohn, 6 <i>Clarke Grove, Sheffield,</i>	1889
295	Dr Herbert Littlejohn, <i>Scarborough,</i>	1892
	Dr W. H. Lowe, <i>Woodcote Lodge, Inner Park, Wimbledon,</i>	
	<i>Surrey,</i>	1845
	Dr Robert Lucas, <i>Dalkeith,</i>	1875
	Dr F. R. Macdonald, <i>Inveraray,</i>	1860
	Dr K. N. Macdonald, <i>Gesto Hospital, Edinbane, Skye,</i>	1880
300	Dr W. B. Macdonald, <i>Port Lodge, Dunbar,</i>	1888
	Dr John A. Macdougall, <i>Canes, France,</i>	1875
	Professor J. M'Fadyean, 101 <i>Great Russell Street, London,</i>	
	<i>W.C.,</i>	1888
	Dr John Mackay, <i>Aberfeldy,</i>	1881
	Dr W. B. Mackay, 23 <i>Castlegate, Berwick-on-Tweed,</i>	1893
305	Professor M'Kendrick, <i>The University, Glasgow,</i>	1870
	Dr James Mackenzie, 66 <i>Bank Parade, Burnley, Lancashire,</i>	
		1894
	Dr W. O. M'Kenzie, D.I.G.H., 37 <i>Belsize Park Gardens,</i>	
	<i>Hampstead, London, N.W.,</i>	1845
	Dr T. J. Maclagan, 9 <i>Cudogan Place, Belgrave Square,</i>	
	<i>London, S.W.,</i>	1875
	Dr Roderick M'Laren, 23 <i>Portland Square, Carlisle,</i>	1882
310	Dr John Macpherson, <i>Stirling District Asylum, Larbert,</i>	1891
	Dr J. W. Martin,	1888
	Dr A. Matthew, <i>Corstorphine,</i>	1882
	Dr J. Moolman, <i>Cape of Good Hope,</i>	1877
	Dr A. E. Morison, <i>Brougham Terrace, Hartlepool,</i>	1889
315	Dr J. Rutherford Morison, 14 <i>Saville Row, Newcastle-on-Tyne,</i>	1882
	Dr J. Ivor Murray, <i>Granby House, St Nicholas Cliff,</i>	
	<i>Scarboro',</i>	1857
	Dr Andrew Scott Myrtle, <i>Harrogate,</i>	1859
	Dr Leith Napier, <i>The General Hospital, Adelaide, South</i>	
	<i>Australia,</i>	1879
	Dr T. Goodall Nasmyth, <i>Cupar-Fife,</i>	1884
320	Dr Ernest F. Neve, <i>Srinagar, Kashmir, India,</i>	1884
	Dr T. Wyld Pairman, <i>H. M. Prison, Lyttelton, New</i>	
	<i>Zealand,</i>	1884
	Professor Bell Pettigrew, <i>St Andrews,</i>	1873
	Dr Alexander Peycr, <i>Zürich,</i>	1893
	Dr J. A. Philip, <i>Rue Victor Hugo, Boulogne-Sur-Mer,</i>	
	<i>France,</i>	1878

		Date of Admission.
325	Professor W. S. Playfair, 31 <i>George Street, Hanover Square, London, W.</i> ,	1857
	Sir W. O. Priestley, 17 <i>Hertford Street, Mayfair, London, W.</i> ,	1854
	Dr J. H. Pringle, 256 <i>Bath Street, Glasgow</i> ,	1886
	Dr S. Hale Puckle, <i>Bishop's Castle</i> ,	1885
	Dr G. M. Robertson, <i>The Asylum, Muirthly, Perthshire</i> ,	1890
330	Dr J. Maxwell Ross, <i>Avenel, Maxwelltown, Dumfries</i> ,	1882
	Dr J. R. Home Ross, <i>Burmah</i> ,	1888
	Dr Joseph C. Ross, <i>Withington</i> ,	1884
	Dr S. Rumbold, <i>Hope Villa, Hillary Place, Leeds</i> ,	1887
	Dr Thomas Russell, 27A <i>Westmuir Street, Parkhead, Glasgow</i> ,	1888
335	Dr Gordon Sanders, <i>Cargilfield, Trinity</i> ,	1892
	Dr F. A. Saunders, <i>Grahamstown, South Africa</i> ,	1884
	Dr Thomas R. Scott, <i>Musselburgh</i> ,	1884
	Dr C. A. E. Sheaf, <i>Toowoomba, Queensland, Australia</i> ,	1871
	Dr G. F. Shiels, 229 <i>George Street, San Francisco</i> ,	1887
340	Dr W. H. Shirreff, <i>Melbourne, Australia</i> ,	1883
	Dr William Simmers, <i>Denburn, Crail</i> ,	1894
	Dr T. Skinner, 6 <i>York Place, Portman Square, London, W.</i> ,	1856
	Dr John Smith, <i>Brycehall, Kirkealdy</i> ,	1889
	Dr W. Ramsay Smith, <i>The General Hospital, Adelaide, South Australia</i> ,	1892
345	Dr Van Someren, <i>Goldhurst Terrace, South Hampstead, London, N.W.</i> ,	1845
	Dr Somerville, <i>Galashiels</i> ,	1877
	Dr A. M. Stalker, 140 <i>Nethergate, Dundee</i> ,	1893
	Dr Graham Steell, 96 <i>Moseley Street, Manchester</i> ,	1877
	Professor Stephenson, <i>University, Aberdeen</i> ,	1861
350	Dr Robert Stirling, 4 <i>Atholl Place, Perth</i> ,	1891
	Dr H. R. Storer, <i>Newport, Rhode Island, U.S.</i> ,	1855
	Dr John Strachan, <i>Dollar</i> ,	1867
	Dr John Struthers, <i>Ngamakwe, Transkei, South Africa</i> ,	1895
	Dr J. F. Sturrock, <i>Arima, Broughty Ferry</i> ,	1887
355	Professor J. Symington, <i>Queen's College, Belfast</i> ,	1878
	Professor Lawson Tait, LL.D., 7 <i>The Creseent, Birmingham</i> ,	1870
	Dr C. Templeman, <i>Sanitary Office, Bell Street, Dundee</i> ,	1891
	Dr Thin, 22 <i>Queen Anne Street, Cavendish Square, London, W.</i> ,	1861
	Dr Alexander Thom, <i>Vicwfield, Creiff</i> ,	1884
360	Dr J. Stitt Thomson, <i>The Mount, Lincoln</i> ,	1877
	Dr J. Batty Tuke, jr., <i>Balgreen, Murrayfield</i> ,	1886
	Dr R. S. Turner, <i>Keith</i> ,	1867
	Dr T. Edgar Underhill, <i>Dunedin, Burnt Green, Worcester-shire</i> ,	1885
	Dr A. B. Urquhart, <i>Murray House, Perth</i> ,	1893
365	Dr Gopal Govind Vatve, care of H.H. the Rajah of Miraj, <i>Bombay, India</i> ,	1895
	Dr A. B. Whitton, <i>Aberehirder</i> ,	1886
	Dr Oswald G. Wood, <i>Indian Army</i> ,	1886
	Dr G. Sims Woodhead, 1 <i>Nightingale Lane, Balham, London, S.W.</i> ,	1883
	Dr Strethill Wright, <i>Manor, Monmouth</i> ,	1871
370	Dr Yellowlees, <i>Gartnavel Asylum, Glasgow</i> ,	1862
	Professor John Young, <i>The University, Glasgow</i> ,	1860

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TRANSACTIONS  
OF  
THE MEDICO-CHIRURGICAL SOCIETY  
OF EDINBURGH,  
FOR SESSION LXXV., 1895-96.

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Meeting I.—November 6, 1895.

Dr WILLIAM CRAIG, *Vice-President, in the Chair.*

I. ELECTION OF MEMBERS.

THE following gentlemen were elected Ordinary Members of the Society:—Dr John Tod, 93 Ferry Road; Dr George T. Beatson, 7 Woodside Crescent, Glasgow; Dr William Henry Miller, 51 Northumberland Street; Dr Donald Macaulay, 108 Lauriston Place.

II. ELECTION OF OFFICE-BEARERS.

THE following gentlemen were elected Office-bearers:—*President*, Dr Argyll Robertson; *Vice-Presidents*, Dr John Wyllie, Dr William Craig, and Dr James Carmichael; *Council*, Drs Burn Murdoch, Byrom Bramwell, Sloan, Dow (Dunfermline), Clouston, A. D. Webster, G. A. Gibson, and H. F. Calder; *Treasurer*, Dr Mackenzie Johnston; *Secretaries*, Mr Hodsdon, Dr Graham Brown; *Editor of Transactions*, Dr Craig.

III. EXHIBITION OF PATIENTS.

1. *Dr Stewart Stirling* showed—(a.) An old man with SKIN ERUPTION. About two and a half months ago the eruption came out first on the breast, and soon spread over the whole of the trunk. There was no itching, but he complained of considerable smarting and pricking. He had always been healthy. He saw him for the first time a fortnight ago, and on a very hasty inspection the eruption appeared as erythematous spots, dark red in colour, raised, and softish to the touch. On pressure with the fingers the redness disappeared. On further examination, after treatment with tar and

calamine lotion, he found a few lichen papules combined with the erythematous inflammation, and thought he had to do with a very rare form of lichen. He showed a drawing in water-colour taken at that stage. The eruption now consisted of numerous small roundish and irregular spots and papules scattered over the trunk, but chiefly on the front and side of the thorax, deep red or purplish in colour. The larger spots were raised, and disappeared on pressure, leaving a brown stain. The spots varied in size from a pin's head to about one-third inch in diameter. The eruption was chiefly confined to the trunk. The arms and legs were free, except at the upper part. One peculiarity was the absence of scales, also the absence of itching. The patient sometimes complained of nausea and want of appetite. On looking up the literature, Dr Stirling said he could not find any case exactly like this. Mr Jonathan Hutchinson had recorded one or two which resembled it, but not completely. Dr Crocker had recorded in about a dozen lines two very rare cases of lichen with crimson red lesions, soft to the touch, and which looked more like erythema than lichen, and which he had called lichen planus erythematosus, but Dr Stirling proposed to call his case by the name of lichen ruber planus erythematosus. The patient was using tar and calamine externally, and taking small doses of arsenic. (b.) A case of LUPUS OF THE SCALP in an elderly man. He had been using mercury and carbolic plaster, which seemed to do very well.

2. *Dr Norman Walker* showed a boy with ERYTHEMA. His two elder sisters also suffered from this form of eruption. The lesion commenced as small brown spots, and spread in a circinate manner. They did not tend to appear in winter more than in summer. This boy had suffered the whole summer. He had excised one of the spots at this early stage, and found, although he had not examined it very fully, the appearance of great cellular proliferation in infiltration around the vessels. He put him first on quinine, following Dr Aliquis's advice, and then Dr Jamieson advised salicylate of soda. As the eruption distinctly got worse under salicylate, he went back to quinine. Dr Aliquis got remarkable cures with 5 grs. of quinine thrice daily, but Dr Walker found it necessary to push the quinine before benefit was obtained. This boy had been under treatment for two months, and was looking very much better, but the hands still remained affected.

#### IV. EXHIBITION OF SPECIMEN.

*Dr James Carmichael* showed a specimen of DOUBLE INTUSSUSCEPTION OF THE INTESTINE of a boy, aged 9, who was brought into hospital with the history of a month's illness. Previously healthy, he was attacked with acute diarrhœa, which reduced him very much. At the end of a week he improved, but afterwards the symptoms recurred, and he was admitted in a very emaciated state.

Shortly after he came in, there was an enormous prolapse of the rectum larger than his (Dr Carmichael's) two fists. It was easily returned, but immediately prolapsed again. The boy was in an extremely grave condition, being practically moribund. There was marked asthenia, feeble pulse, and inability to retain food on the stomach. The tongue was very red and dry, and so far was he advanced in weakness that his mouth was covered with thrush. They did what they could for him. Obstruction was not complete, inasmuch as he passed both flatus and feces to a limited extent, and there was no distension above the first intussusception. He sank in about two days after admission, and they found this very interesting condition of matters,—the ileum invaginated into the cæcum, and this invagination itself invaginated into the transverse colon. On opening the body, they found this mass chiefly situated in the left hypochondrium. The right hypochondrium was so little occupied that one could pass one's finger to the right kidney. During life he could pass his finger easily up to the second intussusception, and could also thus verify the incompleteness of the obstruction. There was no peritonitis. The boy had a subnormal temperature.

*Mr Joseph Bell* said that, by the kindness of Dr Carmichael, he saw the case at a late stage, with a view to operation, but on consideration they felt they could not have got the boy off the table, and did not think it would have been of any benefit. As Dr Carmichael had said, there was no actual stoppage. Altered peristalsis was doing the mischief. There was no adhesion.

#### V. ORIGINAL COMMUNICATION.

A letter from Dr Clouston was read explaining that his medical attendant had advised him that he was not able to occupy the chair, and expressing regret for his absence.

*Dr Gibson* read, in Dr Clouston's absence, the President's

#### VALEDICTORY ADDRESS.

GENTLEMEN,—No member of the Medico-Chirurgical Society now living could, I believe, take the President's chair without a deep sense of his inferiority in mind and attainments to many of the men who have sat there before him. I am no *laudator temporis acti*, but in this I simply state a fact. When I glance down the roll of our past Presidents and see the names of Duncan, Abercrombie, Alison, Christison, Syme, Simpson, Goodsir, and Bennett, my personal feeling—as would be, I believe, that of most of us—is one of profound humility and total unworthiness.

You have been presided over, gentlemen, in the past, not only by great physicians and surgeons, but by great men. We of the present day can only hope most fervidly that the race of giants that lived in those days is not impossible in the future of our

profession. Surely the race that bred these men in the past will again procreate men of might like unto the former generations. I think we are justified, as students of heredity, in anticipating that this will be the case. Brain power seems to come to mankind in waves. In one generation there is an abundance of it, surging up to the highest water-mark of past times, and then again there are low tides of mental energy. If it is any consolation to us, physic is not alone in the trough of the wave at present. Politics, literature, journalism, law, and divinity are all alike crying out for effectual leadership.

Is there no compensation for this state of matters? Nature commonly, when niggardly in one direction, gives some profusion in others. She has a rough way of making up averages, which has been called the "law of compensation in the world." Whether this is always so or not, our self-respect demands that we should feel that we have some good in us, special to ourselves, as individuals and as a generation. Otherwise we should sink into the mental condition of the "submerged masses" among the generations of mankind, and cease to exert even the powers that we possess. If there were in the past a few giants that could have lifted a thousand pounds, there may now exist many fairly strong men who each can be responsible for three hundred. If no one man makes extraordinary advances, are there not ten that are making moderate acts of progress? Does it not require a generation of plodders to discover the applications of the great principles and generalisations which the mighty men of the past have laid down? May not, in fact, the sum total of our work be comparable in its usefulness to mankind to that of the great inventors and the discoverers? Pasteur discovered the part which germs play in disease and health, and Lister applied that knowledge in the antiseptic principle of treating wounds, but to get the full value of the application of both doctrines it is requiring hundreds of hard workers and experimenters in every field, so that suffering humanity may reap the full harvest of health and happiness. In my own department of medicine Conolly laid down new principles, and pointed out the falsity of certain old doctrines and the futility of certain old practices, but it has needed two generations of ordinary asylum physicians, of plain business committees of asylums, and of hard-working Commissioners in Lunacy, so to apply and to extend Conolly's doctrines that those suffering from mental disease should have the full practical benefit of those principles. They are now having that in a way undreamed of by the apostle of non-restraint.

So, gentlemen, in our Society have we not in the last two sessions been doing fairly good work, essential to the progress of medicine and useful to mankind, even though none of us have formulated any far-reaching generalisation or made any epoch-making discovery? Have not Medicine and Surgery been, on the whole, the better for our meetings and discussions? Surely it is not pre-

sumptuous in us to make answer in the affirmative. Surely every member who has given us the result of his experience or thinking, every man who has brought forward a case or shown a pathological preparation, nay, every doctor who has sat on these benches and been willing to learn something from others, surely all these have furthered the work of the Society, and may reasonably expect to get the commendation of the good and faithful servant who is to enter into the joy of the paradise of the dutiful. I do not mean to imply for a moment that the members who have not done any of these things will be driven into the outer darkness where there is no Medico-Chirurgical light. There will, at least, be a place of repentance provided for them during the next two years under my successor Dr Argyll Robertson, where they will be mercifully treated. But beyond a doubt they must show some signs of repentance unto good works. Life is short, art is long. I do most sincerely believe that every man who has joined this Society has a personal responsibility for his membership. He must do some work or some attendances to have any excuse at all. Nature provides the guerdon to every man who earns it by giving that inward satisfaction from duty done which is of great price in every man's inner life. Our muster-roll now runs up to 360 members. What a vast mine of experience does that imply! If every member saw only 400 cases in a year, that would run up to about 15,000 individual cases of disease. To say that there would be nothing new or interesting to others among that vast pathological mass would be to stultify one's self. I venture to suggest that enough is not made out of the cases in ordinary practice for the benefit of the Society, which, it must ever be kept in mind, is primarily composed of, and exists chiefly for the benefit of the general practitioners of medicine. She is the mother of all the other medical societies of Edinburgh, and heartily wishes all her progeny well. But she claims authoritatively that every important thing done in Medicine, Surgery, Obstetrics, and the Specialities shall be brought before her also in a suitable shape. A paper for a special Society naturally goes into more details, is more technical and specialised. When the same subject is brought before us here, it is treated more with relation to medicine or surgery in general, and with more reference to the practical needs of the general practitioners. General physic should know all the chief directions in which specialism is going, and the dominant lines of theory, investigation, and practice in them. All the specialities, on the other hand, should make a point of following the general trend of medicine and surgery. Each should clearly realize the points where they must meet. Thus alone will general medicine and surgery get the full benefit of what the specialists know and can do, and thus alone will the specialists save themselves from falling into dangerous, narrow ruts from which they will not see the great country through which they pass, and of which they are a part. A new modification of a gynecological

operation, an improvement in ophthalmological procedure, a new discovery in the pathology of insanity—I care not what it is—but I say emphatically that the specialist, after describing it in his own Society, is bound to bring it in a suitable form at once before his brethren here. It needs no argument to prove that this is the right course and the wise one for himself and his profession. The Medico-Chirurgical is the only link that here in Edinburgh binds together the whole profession in one, and it is because we all belong to it that specialism has not become a noxious plant with us, and that general medicine has never shown any jealousy or suspicion of it.

An ideal Medico-Chirurgical, with ideal meetings, would cover the whole profession in Edinburgh, and those within easy reach by rail of us. Each meeting would never consist of less than 100 members. There is stimulus and satisfaction in the mere number of your audience, if you have anything to say, which nothing else can give. There should be no minutes except on special occasions. The cases shown should be mostly classified beforehand by a small sub-committee, and so the pathological preparations. Questions should be allowed about cases and specimens, but they should be always very short and very relevant. There should be clinical evenings. The papers should usually be cut down from the form in which they are to be published. The discussions should be short and interesting. Three papers could very well come on each night on most evenings. The gist of everything that is going on should come before us each session. No man should prose or go into unnecessary detail, at the risk of being shut up at once. There should be discussions only now and then, when there is any suitable question to discuss; and it is doubtful whether the speeches should be a series of elaborate exhaustive papers. Every meeting should be so interesting and instructive that members would look on it as a great hardship to be kept away by necessary business. I am a member of half a dozen societies, and I can say, with truth, that there is not a single meeting of one of these that is actually so interesting or so instructive as it could be made if pains were taken beforehand with the papers and the discussions. It is a question whether with us a small committee of the Council should be appointed to hunt up suitable papers and good men with new experiences to relate. In fact, we need a little more organisation in our work. Pray, understand that I am not speaking in any dissatisfied or complaining way. Many of the younger members of the Society have done work of the best kind for us. Many of our older members have shown an example to us all, giving us out of the treasures of their experience things new and old. Any one who says that our discussions on Intra-cranial Surgery and Cardiac Therapeutics were not successful and instructive, either did not hear or read them, or is incapable of forming an opinion. They will both serve



as landmarks that will show exactly the Edinburgh practice and theory on those questions at this time. The wealth of experience which almost all our best surgeons and physicians exhibited to us on these two great questions was, I know, full of instruction to their juniors, and I hope to themselves. The expression of the deliberate and matured opinions of two of the highest original authorities on cardiac therapeutics in the world, Prof. Fraser and Dr Balfour, were of essential service to humanity and to the profession. To hear those, together with the views of almost all the teachers and practitioners of light and leading here, was an education in the subject to many of us, and a great treat to all. The subject is one that concerns every man in practice very frequently. For our profession to have sound and right opinions on the correct treatment of heart affections may make all the difference between comfort or discomfort, pain or freedom from it, work or idleness, life or death to thousands of the world's most useful citizens. I believe that our discussion helped to spread such sound views, and therefore directly added to the sum total of human happiness and work. Could any praise be greater than this? The massage discussion was undoubtedly called for in regard to that practice, and helped to clear the air on many doubtful points of practice.

Prof. Fraser's communication on immunity from snake poisoning was, no doubt, the most original, pregnant, and far-reaching discovery that we had communicated to us, and the Society may well congratulate him, as well as feel proud that the tradition in the profession in Edinburgh is still as strong as ever, that any man who has made a great discovery, or has arrived at the result of any important investigation in Edinburgh, comes at once to the Medico-Chirurgical Society to announce it. We are the natural outlet and channel through which all such advances in medicine here are made known to the world. But to show that new ideas and original researches are often being thought out and carried on by different men, quite independently of each other, Dr A. Calmette of the Pasteur Institute, Lille, addresses me as your President, saying that he had for some time been working on somewhat the same lines as Prof. Fraser to create immunity against snake poisoning. This seems another example of two men a thousand miles apart coming to the same conclusions by the same means, without any sort of communication with each other. In such a case every canon of equitable judgment would give both men an equal share of the credit of the discovery, just as if each had made it, and the other had not.

Any President who has sat in this chair for two years has much reason to be grateful to the members of the Society for placing him in that position; for, in my experience, he has occupied the most instructive vantage-ground that is possible in our profession. He has gained more knowledge, and of a wider kind, and in a more agreeable way, than he could have done in any other position.

I earnestly recommend my younger brothers to covet the President's chair, and get into it as soon as they can. The Society naturally selected as its Presidents for the first sixty-eight years physicians, surgeons, and anatomists. But in 1887 Dr John Smith was elected as a representative of specialism, and I and Dr Argyll Robertson have followed him for the same general reasons. A specialist naturally looks at all things in medicine in some degree from his own standpoint. He asks many questions in his own mind that are suggested by his special point of view. His mind cannot but run in its old grooves. When our pathological friends were showing morbid changes in the organs and tissues, and endeavouring to give local explanations of them, I was often asking myself, What about the influence of the brain cortex and the trophic centres in producing those pathological changes? When the surgeons were discussing the applications of intra-cranial surgery to remove tumours and irritations, suggestions of these methods being dreadfully coarse, considering the delicate tissues they had to do with, and how they must be replaced in time by medical means, were occurring to me. When the methods and effects of massage were being discussed, the question whether the effects, good or evil, that were got were not due to stimulation of the central cortex, through the mechanical effects on the peripheral nerves rather than of the direct effect on the muscles and local tissues, suggested themselves. When cardiac therapeutics were being discussed, one wondered there was so little said about the innervation of the heart muscles, and the relationship of the whole organ to the vascular centres in the cerebral cortex. One could not forget that the emotions are the dominating mental influences, that the old and universal generalisation was that the heart was the seat of the emotions, which is physiologically explainable by the theory that its representative centres in the cortex are closely related to and in the midst of these emotional centres. One could not but apply this theory to explain why morphine, strychnine, and arsenic, and rest and quiet, were so strongly recommended by many of our most experienced physicians in the treatment of heart affections. Those medicines and measures seemed to show that much cardiac therapeutics take place through the brain cortex rather than through its own ganglia and muscular fibres. And one could not help speculating—ignorantly, perhaps—that possibly the good effects of digitalis and strophanthus, especially the latter, were partly nervous.

During the surgical papers and discussions I could not help frequently asking myself, Do the surgeons always and fully realize the import of the power which the brain cortex and mind exercise over their distal subjects, the limbs? I presume there is much room for resistance against many evil things that may happen in every case of wound or injury. There can be no such condition as an entire absence of germs, and, undoubtedly, in the cortex we

must chiefly look for this power of resistance against such threatening enemies, and also the power of promoting tissue repair and general health, which is so all-important.

From the chair, in fact, I was looking on all your proceedings with a mind filled with one idea—if you like. Every case, every opinion expressed, was observed by me from the cortical and mental standpoint. My mode of viewing all things was dominated by the underlying conviction that in the organic hierarchy there was only one king and supreme governor, and that all else—every organ, every function—was more or less servile. The brain cortex was the Pharaoh, whose throne towered above all else, whose will was law, who represented everything, in whom all his subjects centred and ended. Away to the distal ends of the hairs the influence of the cortex is felt, for do not men become white-haired in a night, after receiving a mental shock? But as in Nature and society, the greater the power the greater is the responsibility. The influence, too, is not all on one side. The potentate may be slain or poisoned by the meanest serf. Access, effectual and constant, is provided to the throne for the lowest subject. A few microscopic germs enter from the skin or rectum, and his majesty, the cortex, is fevered, sleepless, irritable, and he then makes the whole kingdom uncomfortable. A bone is broken, and the effect of the pain and shock on the cortex, and, through it, on the whole organism, is instant. What in surgery is commonly called the “system,” should really be called by its true name, the cortex. What gives the outward looks of disease, in face, complexion, and attitude, but the cortex? What is it that so changes the normal working of the facial muscles and eye that we diagnose many diseases and injuries physiognomically? The physician who treats a local inflammation or a fever, and the surgeon who treats a serious injury, without direct and conscious reference to the brain cortex of his patient, is surely not working on physiological lines. This distant but commanding organ, whose irradiant energy is needed for the healing of every wound, and for the callus of every fracture, which forms the nexus with all the rest of the organism, and from which all danger signals are hoisted when things are going wrong, must certainly be reckoned with in every case. Whether we take it into account or not, apart from its trophic energy, its mental relationships count for much in all injuries and wounds. The mental condition of many of your patients is often the most delicate test of the state of the local disease or injury. The mental tissue of the cortex is most delicately responsive to peripheral impressions of every sort. No healing or reparative process goes on well if there is mental depression; and the condition of brain cortex which produces that depression will eventually affect digestion, assimilation, metabolism, and secretion throughout the whole body. And such a disturbed cortical state lessens that most important resisting power against

the innumerable enemies of the organism which are always near. Why do rheumatism and gout, in certain forms, attack the man who is down in nervous energy? Why is the pain of both worst at night, when the cortical state is at zero? Why does cancer develop chiefly as men and women have turned the corner of life, and have entered on the down-grade stage of nervous and mental power? Surely Dr James is right in his supposition that all forms of so-called neoplasm had germs lying nascent in the organism until this ebbing of the resistive and regulative cortical energy left them free to start on their destructive career. What power regulates the relative bulk and status of each tissue constituent of every organ? No organ consists of monoplasm. Its various tissue elements have usually very different degrees of nutrient energy, and require different amounts of blood supply. Take an apparently simple tissue like muscle. What governing force, apart from the brain cortex, so regulates the anabolism and katabolism of the cells so that a certain normal amount and not an excessive store of mechanical energy is always lying in them in a potential form? What prevents undue wasting during great exertion, on the one hand, or pseudo-hypertrophic paralysis during periods of idleness, on the other? Think of the complex functions of an organ like the liver. Who acts policeman to regulate the traffic of blood in different channels, according as its glycosuric or its biliary cells are required, but the cortex and its servants the local ganglia? One can as well conceive an army going on without a general, as the great organs doing their work at the proper times, and in the right way, and according to the organic requirements of the moment, without their master, the cortex, being in command. In the nerve centres themselves, what prevents the connective tissues from overgrowth and sclerosis, but higher nervous regulation?

When we come to think of the action of poisons, such as alcohol and syphilis, why do they attack the vascular and connective tissues in the brain, the kidneys, and the liver, rather than the nerve cells, the glomeruli, and the hepatic cells? Those, we know, suffer, but only secondarily, by pressure and irritation. Is it not because these latter tissues are less innervated from the cortex, and, therefore, less resistive against irritative poisons from within? In looking at a microscopic section of the brain cortex of an old alcoholic patient, nothing is more striking than that the degeneration of tissue, the proliferations, the neoplasms—if there are any such—all affect every constituent of the cortex but the nerve cell and fibre, which seem to have suffered chiefly by pressure and starvation of blood. Why have we far fewer skin diseases appearing on the sensitive finger ends than on the body and limbs, but that the abundant nerve-supply there resists the causes of such diseases? Hypertrophic paralysis, and other forms of muscle degeneration, are

frequent enough in the great muscles of the limbs, with their comparatively few nervous strands; but who ever saw the muscles of expression in the face and eye so affected? They have a nerve-supply, we know, ten times that of the great limbs and body muscles. All those, and many more such considerations, show the enormous effect of innervation, mental and trophic, in resisting disease. No doubt one of the most frequent causes of death itself is that the cortical energy is too low to fight the enemies of life, and not that those enemies are specially strong at the time.

But, gentlemen, I fear I weary you with the iteration of my views as to the supreme and all-pervading influence of the brain and the mind in keeping the organism and all its marvellous interdependent organs and tissues within the fair physiological demesne and out of the pathological morass. But I trust your forbearance will endure that, and much more, from a man who profoundly believes in his specialty, and speaks accordingly.

The worst thing about being president of any society, is the feeling, when your term of office is over, that you "have passed the chair," and the uncheerful suggestions which that feeling brings to the mind. I have always, in other societies, when I had the chance, put off this inevitable day as long as I could for this reason.

The Society has lost several members by death during my term of office. Three of them were such high types of the best qualities of our profession, and I had the great privilege of knowing them all so well, that though their merits have already been worthily told elsewhere, I cannot forbear to throw a stone on their cairns before I close.

No more marked individuality than Dr Thomas Keith ever walked the streets of Edinburgh, or contributed, as he did, so brilliantly to the work of this Society. How well we all can picture his keen, nervous face, which might have been that of a poet, the "far-away" look in his honest eyes, the head set forward, the anxious attitude, and the deep, earnest voice. I had the privilege of often seeing him when he was in Edinburgh; and, out of my line as it was, he once asked me to be present at an ovarian operation. I was glad I went, for I then saw the true psychology of a great operator of the nervous type, who fully realized that every knife-cut, and every stitch, might mean life or death to a fellow-creature whose life was as precious to her as his was to him. Many a time he told me of his sleepless nights and days of anxiety after great operations. It was a fillip to one's moral nature to know him, and a great honour for any man to have his confidence. This Society may well mourn his loss, for it was here he came to communicate the successful results of his first series of ovariectomies, when Edinburgh as yet had had no success in that operation. How many lives he saved since then!

Dr Brakenridge has passed away too. We can all remember his opening of our influenza debate three or four years ago—his

precise, accurate statement of his case, conscientiousness displayed in every tone of his voice, and in every operation of his mind. He was a powerful clinical teacher, a high-minded, earnest man, a subtle, accurate thinker, and a loyal friend. The profession in Edinburgh will never need be ashamed of itself if such as he abounds here.

Dr Thomas A. G. Balfour was a frequent attender of our meetings, and sometimes took part in our debates—often coming here after a hard day's drudgery in general practice. I have known him well since the year 1859, and a better man did not live in our city. With all his strong religious dogmatism, he was full of charity, and full of humour. I always used to tell him he had much of the best of the clerical mind, and some of its defects. Some of his stories were admirable, and his laugh was infectious. He had strong scientific tastes of the old-fashioned observing sort. He was beloved by his many patients, and, beyond a doubt, exerted a powerful influence for good during his long and useful life. He worthily represented those who do the hard work of our profession, and do not always receive an over-abundance of its rewards. If our family doctors were all such as he was, how well we should stand with the public!

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*Mr Joseph Bell* said that he rose, since he had been called upon, but would rather see some one else rise who could better appreciate the address. He thought his friend on the left would have had something to say on the part played by the brain. It was quite right that the specialist should look at things from the point of view of his specialty. He thought Dr Clouston took too pessimistic a view of what would happen twenty or thirty years hence. He (Mr Bell) projected himself into the future, and saw a president delivering his address, and starting very much on the same line. He would say, also, that there were giants in those days, great men in the profession in 1895 or so, and he would mention, as one of the great men who had lived before him, the name of Clouston, because in his own specialty he did not know that there was a greater name than his in Britain. He (Mr Bell) moved a very hearty vote of thanks to their President.

*Dr James Carmichael* said he presumed this motion did not require seconding, as it would be carried by acclamation, but he might be allowed to make one remark. He had listened with pleasure to the address from their talented and versatile President. It contained many suggestions, but he would draw special attention to that one as to the better organization of the Society with a view to perfecting its work. Their Vice-President might take it into consideration whether the Society might not act on that suggestion, and consider the whole matter.

*The Vice-President* said he was sure that Mr Bell had well expressed the feelings of that Society and their appreciation of

this very admirable address by their President, Dr Clouston, and in conveying this vote of thanks, which he presumed was carried unanimously, they expressed their sincere regret at his illness, and sincere wish for his speedy and perfect recovery.

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### Meeting II.—December 4, 1895.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. ELECTION OF MEMBERS.

THE following gentlemen were elected Ordinary Members of the Society:—A. F. Wood, M.B., City Hospital; R. W. Inkster, M.D., 38 Montgomery Street; Andrew Balfour, M.B. C.M., 51 George Square; W. Leslie Mackenzie, M.A., M.D., Medical Officer of Health, Leith; John A. H. Duncan, M.B. C.M., 32 Morningside Drive.

#### II. EXHIBITION OF PATIENTS.

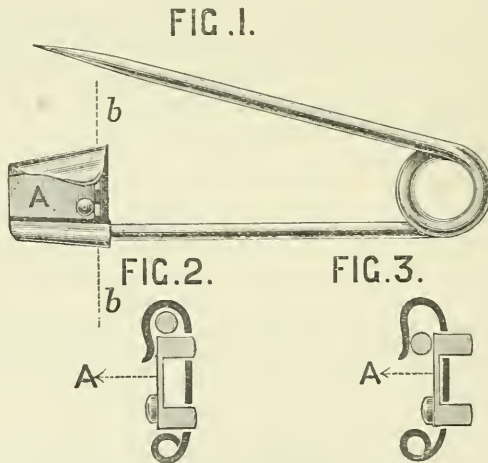
1. *Dr R. A. Lundie* described a boy, aged 13, who was brought under his notice about a year ago with a peculiar TUMOUR OF THE UPPER LIP. Dr Lundie showed photographs which, he said, gave a good idea of the appearance, especially the profile one, in which the patient looked as if he had a supernumerary nose or parrot's beak. It was a firm tumour, about equally distant from skin on outside and mucous membrane on inside. It was first noticed six months before, when about the size of a pea. It had grown steadily, at first slowly, later more rapidly. It was exactly in the middle line. Twelve months had elapsed since removal, and there was no recurrence. He showed the tumour. A wedge had been removed from the front below the skin surface for microscopic examination. Mr Stiles examined it, and reported that it was an endothelioma undergoing myxomatous degeneration, with here and there cartilaginous development. Sections of the tumour were shown under the microscope.

2. *Dr Allan Jamieson* showed a case of MORPHŒA, or circumscribed scleroderma, in which there was as yet no lardaceous deposit. Richard Law, aged 10 years, 73 Albert Street, was admitted to Ward 38, Royal Infirmary, on 22nd October 1895. He was well grown and healthy. Sixteen months before, a small pink spot appeared under the right eye, and gradually increased in size. Other patches had shown themselves since. They were all of a violaceous tint, that under the eye being the deepest in colour. It extended from one side to the other of the lower eyelid, and was about half an inch deep. It faded

entirely, but momentarily, on pressure. There were two rounded patches, the size of a shilling and a sixpence respectively, on the right side of the chin; two slight ones on the right side of the upper lip, one on the tragus of the ear of the same side, and there was a faint violaceous band stretching from thence along the jaw, losing itself imperceptibly in the cheek. All these appeared in the summer of this year. In one of the patches on the chin there was a brown pigmentary spot. There was no thickening of the skin, nor were there any subjective sensations. There was considerable thickening of the inferior turbinated bone, which was twice cauterised by Dr M'Bride. All the lesions were restricted to the area of skin supplied by the second and third branches of the fifth nerve. The only condition which at all resembled this was morphea of a very superficial type, and similar patches had come under his notice associated with the ordinary ivory patches elsewhere. The full name and address of this boy are given to facilitate reference should the patches develop further in future.

### III. EXHIBITION OF INSTRUMENTS.

1. *Mr Hodsdon* showed, for *Dr Felkin*, a new form of SAFETY-PIN, which he thought would meet with the approval of the mem-



*A*, Fig. 1, is a flat spring. Its forward end is bent at right angles with length of pin at line *b b*. This part forms the lock, and is shown in Fig. 2 with pin in catch. In Fig. 3 it is shown pressed aside, with pin passing inward or outward.

bers of the Society. He thought it avoided all the disadvantages of the ordinary safety-pins now in use. It was invented by *Mr Robb* of *Kincairdine O'Neil*. To open the pin, simply press the little knob, which at once releases the sharp arm.

2. *Mr Stiles* showed a TRUSS FOR INFANTS, which he thought was an improvement on the trusses usually employed. A most



useful truss in simple cases was the ordinary skein of worsted. It was comfortable, and the mother could take it off every two or three days and easily replace it. If the hernia was large, and the inguinal canal more patent than usual, it sometimes happened that the worsted truss did not suffice to keep up the rupture. In these circumstances they generally sent the patient to the instrument maker, and got him to make an ordinary spiral truss. In his experience, whenever the worsted truss failed, the ordinary spiral spring truss was almost sure to fail also. Great objections to the spiral truss were that an aggravated hernia required a strong spring to keep it up. After the child had worn it for a few days, it was found that a good deal of irritation was produced over the inguinal ring, and the truss was put aside by the mother on account of the pain it gave to the child. An infant would grow out of a truss in about three months. In the case he was about to show the child was circumcised, and an ordinary spiral truss ordered. The child began to wear it when seven weeks old. It soon became too small. A second truss, which the child wore for three or four months, failed to keep up the hernia satisfactorily. He had recommended a radical cure; but his friend Dr Hartley informed him about the pneumatic rubber truss, which often succeeded when the worsted truss failed. It consisted of a horseshoe-shaped air-bag placed around root of penis and scrotum with its concavity downwards, and attached below and around the pelvis with a rubber band. This truss had proved very satisfactory, having kept up the hernia continuously for a month.

#### IV. ORIGINAL COMMUNICATIONS.

##### 1. OXALURIA AND THE EXCRETION OF OXALIC ACID IN URINE—AN ABSTRACT.

By JAMES C. DUNLOP, M.D., F.R.C.P. Ed., M.R.C.S. Eng.

THIS paper is an abstract account of an investigation which I have carried on in the Laboratory of the Royal College of Physicians, and which is described at length in the *Journal of Pathology and Bacteriology*, vol. iii. For previous literature, methods, and details of observations I refer to that journal. I venture to read this before this Society, as it is a subject with an application in clinical medicine.

The previous literature is extensive. The earliest writings are almost entirely confined to the description of oxalate of lime as found in urinary calculi. Later, the introduction of the use of the microscope into clinical medicine led to the recognition of the crystals of oxalate of lime, and of their frequency in the urine. This was soon followed by papers showing how a deposition of these crystals was frequently associated with certain disorders, especially certain disorders of the digestive system, and these led up to the

recognition of that disordered state of the metabolism known as oxaluria. Chemical analysis of urine to study the excretion of oxalic acid is more recent, and has been adopted by physiological investigators only, being too long and tedious for purposes of clinical medicine.

These chemical and clinical observations referred to are of a most contradictory kind, and at present one is unable to make any inferences with certainty from the recognition of crystals of oxalate in urine.

My investigations were facilitated in three ways.

1. By having at my disposal a centrifugal machine, which enabled me to examine in a thorough manner the deposit of urine.

2. By adopting Dr Reoch's suggestion of adding alcohol to urine as a qualitative test for the presence of oxalic acid; for if oxalic acid is present in a urine, and alcohol be added, then there is always a precipitation of crystals of oxalate of lime.

3. By devising a new, and I trust accurate, method of estimating the amount of oxalic acid in urine.

In the earlier observations I used what is the generally accepted method of estimation, that of Neubauer, but with this the results were so uncertain and so contradictory that I was unable to formulate conclusions.

I shall consider, first, the frequency of excretion of oxalic acid in urine; second, the precipitation of oxalate of lime in urine; third, the source of the oxalic acid; fourth, the amount excreted, and the cause of its variation; and fifth, the clinical condition called oxaluria.

1. *Frequency of Excretion of Oxalic Acid.*—Observation with the microscope shows that oxalic acid is frequently present in urine; this has been known for long. More recently the chemical analyses of Fürbringer and others have shown that it is almost constantly present; but as these analyses are very tedious, and the number of urines examined must necessarily be limited, such a general statement as that oxalic acid is constantly present can scarcely be concluded from them. I have examined a large number of urines qualitatively by Reoch's method, and as the result through the whole series was constant, I conclude that the urine of all men eating an ordinary mixed diet contains oxalic acid, and that consequently *oxalic acid is a normal and constant constituent of human urine.*

2. *Precipitation.*—Oxalic acid is found precipitated in urine as the calcium salt. This is generally described as appearing in two varieties of crystals, octohedral and dumb-bell.

That the octohedral crystals are genuine crystals of oxalate of lime is readily demonstrated; in urine they have the same solubilities as artificial crystals, and similar crystals can readily be produced by adding a soluble lime salt to a solution of oxalic acid.

Dumb-bell crystals essentially differ from these, as they cannot be artificially produced from pure solution of a lime salt and oxalic acid, and do not have the same solubilities as the artificial octohedral crystals, being soluble in acetic acid, and their solution being accompanied by effervescence. This last fact shows that these dumb-bell crystals are not oxalate crystals at all, but are crystals of carbonate.

The frequency of the occurrence of crystals of oxalate of lime in healthy urines has been studied and noted by Walshe, Galois, Bacon, and Smoler, and these four observers state the percentage of urines in which it occurs as 28, 36, 41, and 57. My own observations show this percentage to be 35. From these figures one can conclude that this precipitation occurs in at least one out of every three normal urines.

What the essential difference between urines depositing and those not depositing crystals of oxalate of lime is, has not previously been shown. All normal urines contain oxalic acid, and they all contain lime, which, of course, tends to precipitate it. Previous arguments and my own observations show that this precipitation is prevented partially or entirely by the presence of acid phosphate of soda, and possibly by the presence of other solvents. The difference between urines precipitating and those not precipitating may be either an excess of lime present, an excess of oxalic acid present, or a diminution of the solvent action of the urine.

That the precipitation is not due to variation in the amount of lime in the urine is evident from the fact that in all urines there is an excess of lime. This can readily be demonstrated by adding oxalic acid to any specimen of urine, as this always determines a precipitation of oxalate of lime crystals.

The earlier writers, without any direct observation, considered the precipitation as indicating an excessive excretion of oxalic acid, but this has more recently been denied by Fürbringer, who made a long series of estimations.

My observations do not corroborate the conclusion of Fürbringer, for on comparing the percentage of oxalic acid present in urines depositing and those not depositing oxalate of lime crystals I find a marked difference. In urines depositing the crystals I have found the average percentage of oxalic acid to be  $\cdot 0025$ , while in those not depositing the crystals it is  $\cdot 0010$ . My conclusion, therefore, is that a deposition of crystals of oxalate of lime shows an increased percentage of oxalic acid, and that if the total quantity of urine passed in twenty-four hours be of an average amount—over 1500 c. c.—then a precipitation indicates an increased excretion.

Whether or not variation of the solvent action of urine affects the frequency of this precipitation I cannot definitely say, but my observations show it is not such an important factor as the per-

centage of oxalic acid present. I gather this from the fact that out of a series of urines examined, nearly all of them with over .0016 per cent. of oxalic acid deposited the crystals, and nearly all with less did not do so. The overlapping of the figures was so small that the effect of the variation of the solvent action cannot be great.

3. *Source of the Oxalic Acid.*—Earlier writers observed that eating certain vegetables determined a precipitation of oxalate of lime in the urine, and from this concluded that the food stuffs were the source of the oxalic acid. More recently, various theories have been advanced to show that oxalic acid might be produced in the metabolic changes of the proteids and carbohydrates, and the source at present is considered to be uncertain.

To investigate this point I examined the urines of patients who were restricted to a diet which was free from oxalic acid, as is the case when the diet is restricted to milk, and I found that this diet, if long enough continued, arrested the excretion of oxalic acid, and consequently conclude that the *oxalic acid in the food stuffs is the source of the oxalic acid in urine.* To corroborate this I carried my observations a stage further, by, after having observed the effect of the milk diet on patients and on myself, adding to it some tea, and in each instance this addition of tea, which contains oxalic acid, determined an excretion of oxalic acid in the urine.

4. *Amount of Daily Excretion and Factors influencing it.*—To state accurately what is the average daily excretion of oxalic acid is difficult, and is so because the daily quantity varies considerably, and also because the analyses are so long and tedious, each occupying about ten days, that the series from which the calculations are made are too short to give definite results.

Fürbringer, who used Neubauer's method of analysis, states it as being "something under .020 grm.;" he does not commit himself to an exact figure, but from his published figures, using only those previous to the administration of a drug, I calculate his average to be .0055 grm.

From a series of thirty-five estimations I have found that the majority of urines contain between .010 grm. and .025 grm., and that the average of these was .0172 grm. These estimations were made with my own method.

During my investigations I have found and studied two factors influencing the excretion of oxalic acid—these (1), the amount of oxalic acid swallowed; and (2), the acidity of the stomach contents.

(1.) That the amount of oxalic acid swallowed can do so is shown by the increased excretion which follows the administration of a soluble salt of oxalic acid. In one experiment, administration of potassium oxalate increased the daily excretion of oxalic acid from .010 grm. to .026 grm., and in another experiment from .011 grm. to .034 grm.

(2.) Acidity of the stomach contents. In the stomach the oxalic

acid swallowed is in contact with lime salts derived from food and drinking water tending to precipitate it, and with acids, hydrochloric and lactic, tending to dissolve it, and thus render it in a condition favouring its absorption. In five cases I administered hydrochloric acid in sufficiently large doses to increase the acidity of the stomach, and in every case the average daily excretion of oxalic acid was increased by it; the average increases produced were  $\cdot 0215$ ,  $\cdot 0095$ ,  $\cdot 0199$ ,  $\cdot 0129$ , and  $\cdot 0100$  gm. Lactic acid was twice given, and had a similar action, the average increase caused by it being  $\cdot 010$  and  $\cdot 0021$  gm.

That these two factors influence the excretion in the way they do corroborates the theory of the source of the oxalic acid in urine which I have supported.

5. "*Oxaluria*."—By oxaluria I refer to the clinical condition described by Dr Begbie, not merely a subject whose urine deposits crystals of oxalate of lime, but one who, in addition to this, has the well-known combination of symptoms associated with the name oxaluria.

The symptoms of "oxaluria" are of four kinds,—(1.) Altered condition of urine. (2.) Pains. (3.) Dyspepsia. (4.) Nervous symptoms.

*Condition of Urine*.—There is only one constant change noted in the urine of all published cases, and that, of course, is the deposition of crystals of oxalate of lime. That this symptom alone is insufficient to enable one to diagnose any disordered condition of the system is evident from the frequency of the occurrence of these crystals in the urine of healthy subjects,—more than one urine out of three of healthy subjects depositing them. As stated earlier in this paper, I consider that a deposition of these crystals indicates an increased excretion of oxalic acid. But to what this increased excretion is due is not indicated by an examination of the urine.

*Pains*.—The principal ones are headache, pains in the region of the stomach, and pains in the lumbar region. The first two I shall consider when discussing the dyspeptic symptoms. The pains in the lumbar region are usually attributed to mechanical irritation set up by the passage of the crystals through the pelvis of the kidney and ureter; and with this I agree, as the crystals, though small, have exceedingly sharp angles, and observations on cholera patients, where the flow of urine is arrested or very much retarded, show that the deposition can take place as high up as in the urinary tubules. The pains in the lumbar region, then, give no indication of the pathology of the condition.

*Dyspepsia*.—This is a constant symptom in oxaluria, and is, I believe, the principal one. I do so for three reasons,—*First*, because in acid dyspepsia symptoms almost identical to those of "oxaluria" occur. In both there are pains in the region of the stomach, a feeling of fulness, acid eructations, and so on; and what is more conclusive is that such an authority as Hayem—

*Leçons de Thérapeutique*—in describing acid dyspepsia describes as symptoms of that the typical pains met with in oxaluria, pains in the back, the sides, and the lower part of the lumbar region, and also describes the nervous symptoms similar to what are met with in “oxaluria.” The symptomatology of the two conditions are so similar that, without examination of the urine, they cannot be distinguished. *Second*, because both acid dyspepsia and oxaluria are treated and relieved by the same means. Both are treated either by the administration of acid before food or by alkali after food. *Third*, because an artificially produced hyperacid condition of the stomach causes an increased excretion of oxalic acid. I have alluded to this earlier in this paper, when considering the factors which influence the excretion of oxalic acid. In my experiments acids were given in sufficiently large doses to increase the acidity of the stomach contents, and these markedly increased the excretion of oxalic acid.

That the increased excretion which occurs in oxaluria is due to increased absorption of oxalic acid contained in the food stuffs, and not from any other source, is shown by the fact that in oxaluria its excretion is arrested, or at all events greatly diminished, by restricting the patient’s diet to a pure animal one. This has been demonstrated by Cantani; he adopted animal diet as a means of treatment, and by it alone freed the urine of oxalate crystals and gave relief from the lumbar pains.

Whether acid dyspepsia is a derangement of the stomach, as taught by Hayem, or a neurosis, as taught by Ewald, is immaterial in discussing the similarity of oxaluria and acid dyspepsia, for what can be the etiology of one can also be the etiology of the other.

*Nervous symptoms* met with in oxaluria might be explained in two ways,—by being due to an excess of oxalic acid in the system, or by being due to the dyspepsia which is present. I believe that it is to the latter that they are due, and do so because of their similarity. In both there is a similar headache, a neurasthenia, an irregularity of sleep, etc., so whatever can produce these in acid dyspepsia can also do so in “oxaluria.” That they are not due to an accumulation of oxalic acid in the system is made evident by a consideration of the quantity of oxalic acid which is required to produce nervous symptoms, and comparing it to the quantity excreted in urine. Esbach, experimenting on himself, swallowed over two grammes of oxalic acid, and excreted during the following twenty-four hours 181 gm.,—a quantity many times greater than what is found in urines depositing crystals of oxalate of lime, and he experienced no nervous phenomena at all.

These considerations of the symptoms of oxaluria show that it does not essentially differ from acid dyspepsia, excepting, perhaps, that the deposition of oxalate of lime in the urine occurs with more regularity. Crystals of oxalate of lime are frequently seen in

healthy urine and in the urine of patients suffering from very varied diseases, and indicate no special disorder of the metabolism, consequently there are no grounds for separating oxaluria from other forms of acid dyspepsia. Patients who suffer from that combination of symptoms to which the name of oxaluria has been applied, whether their urines deposit oxalates or do not, should be recognised as suffering from acid dyspepsia, and should be treated accordingly.

Oxaluria is no pathological entity, and the recognition of oxalates in a urinary deposit has no diagnostic value.

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*The President* expressed the indebtedness of the Society to Dr Dunlop for his interesting account of his careful researches, comprising, as it did, an account of the symptoms associated with oxaluria, the sources of the oxalates, and the forms in which they appeared in the urine.

*Dr Noël Paton* said he spoke with some diffidence, not being a physician, at least not for the last six years; but he had taken a great deal of interest in the subject, owing to its bearing on the general chemistry of the body. The work had been done in the College of Physicians' Laboratory, and therefore he had had an opportunity of watching it in its progress. He thought the President had rightly expressed the indebtedness of the Society to Dr Dunlop for the very painstaking character of this research. It was difficult, perhaps, for the ordinary clinical physician to appreciate all the difficulties he had had to tackle. It had extended over two or three years, and he had met with many difficulties, and overcome them in an admirable manner,—*e.g.* the difficulty in separating the oxalic acid, and the difficulties arising from the imperfection of previous methods. There could be no doubt that he had shown quite clearly that the presence of oxalates in the urine was not due, as was still thought and still taught by many, to metabolic changes, changes of proteids, carbohydrates, and fats, as they occurred in the body. It was a point of very great importance that he had shown that these oxalates really came from the oxalates of the food. He had, in his (Dr Paton's) opinion, succeeded in exploding one of those clinical bubbles that were apt to last for many generations on account of the credulity of physicians. He meant to say that physicians were apt to accept the authority of such an observer as Begbie; and the admirable description he gave of the condition had predisposed physicians to make their cases fit his description rather than to investigate cases on their own basis. He believed most of the cases of oxaluria described had been cases of acid dyspepsia. Physicians had looked for and found oxalates, and called the case Begbie's oxaluria. Dr Dunlop had shown clearly that the symptoms were not due to oxalic acid poisoning, but were merely the symptoms of acid dyspepsia. He (Dr Paton) thought it a matter of great regret that in a School

like their own more work of this kind was not done. Why should all the work on the diseases of metabolism come from Germany? Was it because their own men were taught to accept authority, or because their scientific training was not thorough? With more painstaking observations of this kind many advances in clinical medicine might be made. It was creditable that such a piece of work should have come from their Laboratory.

*Dr Graham Brown* said that he was sure they must all join in the congratulations that had been given to Dr Dunlop for the very admirable paper he had read them. He had put the case very strongly against oxaluria. Possessing, as he (Dr Brown) did, the natural credulity of the physician, he was disposed to stand up for his old friend oxaluria, and he did it on three grounds,—(1st), the congeries of symptoms Dr Dunlop had so admirably described; (2nd), that undoubted cases had occurred in which oxaluria and diabetes had alternated in the same case; and (3rd), on the ground, which was less satisfactory, but which was very interesting to himself, of certain recent experiments, where, in the case of dogs, respiration had been more or less interfered with by means of Sayre's applications to the thorax and abdomen; and where impeded respiration had been exactly followed by a great increase of oxalates in the urine. As the dog got more and more accustomed to its unusual respiratory position dyspnoea diminished, and so did the excretion of oxalates, which had reached its acme during the acme of the dyspnoea. He had not gone into the subject carefully; but he had always considered oxalate of lime in the body in these cases as being one of those intermediate products of metabolism which only appeared to a very slight extent in the urine in the form of oxalates, but the greater part of which underwent certain other changes, resulting chiefly in the breaking up into carbonic acid and water. If one's old theories were correct, it would be extremely interesting, in view of that experiment on dogs. These were the three main reasons that occurred to him at the moment for supporting oxaluria. He supposed there were others.

*Dr Gillespie* said that Dr Dunlop by giving vegetables, or by giving hydrochloric acid or other acids, increased the excretion of oxalates, or by giving a meat diet he could decrease it. He thought Dr Dunlop was rather dogmatic in his statement that in no other circumstances could they get oxalates in the urine. They knew that in Italy oxaluria was very common, and most of the work on oxaluria had been done in Italy, because the peasants there were largely vegetable-eating. In Germany they ate less vegetables. According to his reminiscences of Germany, the vegetables formed a comparatively small part of the diet. The German physicians did not recognise oxaluria at all (as a disease), and they stated that it was very uncommon there. Here it was fairly common, and also in America, according to Osler. Diet, therefore, must have something to do with it. A largely vegetable diet gave rise to considerable



fermentation both in stomach and intestines. With a meat diet there was much less fermentation either in stomach or intestines. One German observer, Albu, ascribed oxaluria chiefly to intestinal fermentation, and, in fact, regarded it as one of the class of diseases known as auto-intoxications. Dr Dunlop said the symptoms were almost synonymous with those of acid dyspepsia, but he forgot to say which acid dyspepsia he meant. If he had said hydrochloric acid dyspepsia, they might have understood; but, on the other hand, they had lactic and acetic acid dyspepsia, in which hydrochloric acid was diminished. With increased hydrochloric acid they had the urine less acid; with diminution of hydrochloric acid and increase of the other acids they had the urine more acid. The symptoms of these two were quite different. What Dr Dunlop described was rather the acetic and lactic acid dyspepsia. Oxalic acid was a very simple body, and was formed artificially by the action of nitric and other acids on molasses and sugars. It might easily be formed from the starches, which were changed into sugars in the intestinal canal by the action of some of the acids there. Sugar might, by means of bacterial action, be transformed into oxalic acid. One of the German observers had maintained that proteids could be transformed into oxalic acid. There was also the action of cyanogen with water. From the ease, therefore, with which oxalic acid could be formed in the intestine, there might be fallacies affecting Dr Dunlop's work. Of course, all these criticisms had nothing to do with the fact that he could play on the oxalates in urine as he liked, increasing or diminishing them. The oxalates, however, might have other sources than those mentioned by him.

*Dr James* said that he had not intended speaking that night, but felt that he was bound to take up the cudgels in this matter. In several respects he might take up a position a little opposed to Dr Paton. Dr Paton had referred to the great difficulty of the research of which Dr Dunlop had given an account. Physicians knew another difficulty which Dr Paton had not mentioned, viz., getting patients to attend to instructions as to diet. The research was a very important one scientifically, and also from the clinical point of view, and they were all much indebted to Dr Dunlop. As a physician, however, he thought, with Dr Graham Brown and Dr Gillespie, that there might be sources of fallacy in connexion with the results. Was it the case that oxalates were derived entirely from the food, and in no case the result of tissue metabolism? He was one of those who still believed that they might be the result of tissue metabolism. They knew the close connexion of oxalic acid with uric and carbonic acid, and with fat, sugar, proteids, and so on, and that led them to believe that out of tissue metabolism they might have oxalates arising. It was true that with an animal diet or milk diet oxalates did not appear in the urine. But with animal diet or mixed diet it was quite possible

that the dyspepsia, that faulty metabolism which might result in oxalates being formed, was got rid of. He would like Dr Dunlop to put an individual on milk diet for several days, and then induce in that individual acid dyspepsia. It would only be when he heard that that did not induce oxalates to appear in the urine that he would be disposed to accept entirely what they had been told that night.

*Dr Stockman* said that he thought Dr Dunlop had in one way fully proved his thesis,—namely, that taking a healthy man,—*e.g.* himself or some patient in the Infirmary,—he managed, by giving certain diets containing a quantity of oxalates, or no oxalates at all, to free the urine from oxalate of lime, or to increase the quantity just as he pleased. Like Dr Graham Brown and Dr James, he doubted if Dr Dunlop was quite correct as to oxalic acid not being formed from sugar, starches, and such things in the intestinal canal. They all knew that oxalic acid was very easily formed outside the body from many articles of food. The point was that certain persons were said to excrete a large amount of oxalates in certain conditions. Dr Dunlop apparently had failed to get hold of a person of that kind, who could convert certain kinds of food into oxalates, and who suffered accordingly. Were he to make observations on a large number of people, he might come across a case which, on certain diets, would produce oxalates, and have low spirits and all the other symptoms he described. It was difficult to pitch on a number of cases so as to get positive results. Negative results were easy enough to get. He (Dr Stockman) thought far too much stress was laid on the presence of oxalate of lime crystals in the urine. With regard to what Dr Paton said about the physician he did not quite agree; but the microscopic examination of the urine for oxalate of lime crystals was quite insufficient to show the amount of oxalic acid in the urine. Dr Dunlop had told them there was always a certain quantity of oxalic acid, and that it was only deposited under certain conditions. Under certain conditions of the urine oxalate of lime crystals came down, and under others oxalate of lime remained in solution. There might be physicians who subjected the urine to quantitative chemical examination for oxalic acid, but hitherto he had not known them. They made a microscopic examination, which was absolutely no test. There might be oxalic acid present, or there might not be. The microscopic test merely showed that there was something in the urine which threw it down. A body which was present in 35 to 40 per cent. of all urines examined could not have any great significance as a symptom of disease. As a matter of fact, he found that normally there was an excretion of very small quantities of oxalic acid in a healthy man. Very often 2 or 3 grs. were given in cystitis and bladder conditions without, so far as he knew, producing any of the symptoms. So far as his experience went, he

had never come across a case of oxaluria in the sense in which it had been understood by English physicians.

*Dr Leith* said he should like to say a few words on this question, although not specially concerned in it. He had followed *Dr Dunlop's* thesis with great care, and thought that he had exploded, he would not say the bubble, but rather the remains of the bubble, for he thought *Dr Paton* hardly put it correctly, and that he hardly gave the physicians the credit due to them. It was, of course, the work of *Dr Begbie* of their own town, also especially of *Dr Golding Bird* of England, who brought this question before them so very strongly, and drew apparently a perfect and complete picture of dyspepsia along with certain nervous symptoms and hypochondriasis, which apparently made a very good picture of the disease, and impressed physicians so much that for a long time they were inclined to regard the identity of the disease as established. The careful work of *Beale*, *W. Roberts*, etc., had practically upset this view, and he rather thought that *Dr Dunlop* had but dissipated the remains of the bubble. He thought the other questions as to the possible conversion of proteids into oxalic acid were extremely important; and it would be to *Dr Dunlop's* credit if the belief, which seemed to them natural and reasonable, that proteids were readily converted into urea and uric acid, and thus converted into oxalates, should turn out to be false. Perhaps *Dr Dunlop* required to do a little more before he thoroughly proved his thesis. He gathered from *Dr Dunlop* that the oxalic acid was excreted in the form in which it existed in the food. According to the tables, the amount taken was in excess of that excreted, so that some of it was absorbed. *Dr Dunlop* might investigate cases of a phtisical nature or of chronic cardiac disease. These cases were shown to have sometimes a greatly increased excretion of oxalic acid, as indeed also occurred generally in cases where imperfect assimilation and mal-oxidation were going on. The increased amount of oxalic acid was found to do no harm whatever. A greater amount had been found in phtisis and in some cases of chronic heart disease than in cases of so-called "oxaluria." Perhaps the examination of such cases might throw further light on the question.

*Dr William Russell* said he would like to add his word of praise to the words already spoken in commendation of *Dr Dunlop's* work. He was a somewhat credulous physician, but he was not sufficiently credulous to accept all laboratory work as a guide to the physician. He thought, however, *Dr Dunlop* had proved his thesis in so far as he could play on the oxalic acid secreted, and he had proved that it was very largely derived from vegetable food; but it seemed to him (*Dr Russell*) that there had been various misconceptions shown on this subject. In some of the recent works on medicine, some books even in the hands of students, they found merely a passing reference to oxaluria. It was largely exploded as a morbid entity. Then, further, when they were taught that

there was a condition of oxaluria to which they ought to pay attention, it was also impressed on them that this oxaluria was associated with various gastro-intestinal disorders, with dyspepsias of various kinds; and this seemed to him the turning-point of the whole question, viz., first, whether in all acid dyspepsia there is a large deposit of oxalate of lime; whether there was not an abundant increase—he would not say of oxalic acid, because there was no satisfactory method of estimating it until Dr Dunlop introduced this admirable method of his own—of crystals of oxalate. These were not present in all bad acid dyspepsias, so that there must be special forms of disorders in which this great increase of oxalic acid was present, which was so prominent that it had led physicians to regard it as so far an entity, because associated with other very marked and somewhat characteristic symptoms. He thought the question which Dr James had submitted to Dr Dunlop was really a somewhat important one, viz., whether on a diet free from vegetables he might not be able to produce oxalic acid in the urine; and whether he could really exclude all the substances that had been mentioned as possible sources of oxalic acid, because he thought that in the present state of their knowledge it was exceedingly difficult to exclude all these processes from the gastro-intestinal tract when dyspepsia was present.

*Dr Gillespie* said that Dr Leith seemed to have taken him up wrongly as to proteids. He argued that a proteid diet was one which largely obviated and prevented sepsis or fermentation in the intestines, and might therefore diminish oxalates in the urine.

*Dr Dunlop* said he must first thank the Society for the very kind hearing and full discussion they had given his paper. Dr Graham Brown, after speaking of the symptoms, had mentioned the experiments of Reale and Bocri with Sayre's jackets. He (Dr Dunlop) had only seen an account of it occupying about half a page of a small journal, in which neither exact methods nor amounts were stated. A possible explanation was that the increased amount of oxalic acid was due to congestion of the alimentary tract, caused by dyspnoea, inducing increased absorption of oxalic acid already present. There was no evidence that oxalic acid was oxidised in the body. All the oxalic acid taken did not appear in the urine, simply because a great deal passed out in the faeces, which he had not analysed owing to the difficulty of the work. No amount of dyspnoea or interference with respiration had ever caused an accumulation of oxalic acid in the body; if it were so, oxalate of lime would have been produced, and solid oxalate of lime has never been found in the body. Dr Gillespie's view was that oxalic acid was produced by decomposition in the intestines. He (Dr Dunlop) considered that his experiment with milk diet quite proved the contrary. A milk diet stopped oxalic acid. Tea was given, and oxalic acid appeared. As regards the acidity of the urine in acid dyspepsia, well, all the statements which had been

made were made from a percentage examination which was valueless. He must thank Dr James for putting patients at his disposal in the hospital. As to the further test proposed by Dr James of inducing acid dyspepsia after several days' milk diet, he was looking forward to an opportunity of applying it in Dr James's wards. Dr Stockman had spoken of finding extraordinary patients. He (Dr Dunlop) had not been able to do so. To give one an idea of the rarity of the condition, there had only been three cases of oxaluria in the Infirmary in the last three years. Acid phosphate of sodium did not precipitate oxalates, but was a solvent of them. Precipitation was due to increased percentage of oxalic acid present. Dr Leith had referred to Dr Golding Bird, who certainly gave a very full description of it; but the man who succeeded in bringing it prominently before the notice of the profession was Dr Begbie. Oxalic acid had been prepared from uric acid but not from urea. He (the speaker) believed that the amount of absorption was one of the principal factors affecting the amount of excretion. Dr Leith talked of the increase of oxalic acid in phthisis. Dr Cantani, of Naples, had worked at that point, and by a very large number of observations had shown that it was not so. He (Dr Dunlop) would certainly expect that a very large proportion of acid dyspeptics would deposit crystals of oxalate in the urine.

## 2. REMARKS ON THE RESULTS OF SURGICAL MEASURES IN A SERIES OF CEREBRAL CASES.

By G. A. GIBSON, M.D., F.R.C.P. Ed., D.Sc., Assistant-Physician to the Royal Infirmary; Lecturer on Practice of Medicine, School of Medicine, Edinburgh.

DURING the last four years many examples of organic disease of the brain have been under my care, and of these cases a certain number have presented symptoms demanding operative measures. The opportunities thus afforded me of observing the symptoms, and considering the treatment, have led me to form certain conclusions, which it is my aim to state as briefly as possible in this paper, after casting a rapid glance at the series of cases treated by surgical means.

### I. CEREBRAL TUMOUR WITH JACKSONIAN SYMPTOMS.

*(Reported by Dr Gerald Fitzgerald.)*

A. A., a slater and chimney-sweep, aged 43, married, presented himself before me in the medical waiting-room, January 19, 1891, complaining of persistent headache, with loss of power in the left arm and leg.

The patient had no hereditary tendencies to disease, and seemed to have no specific taint. He had been married for many years, and was the father of thirteen children; five of them died in early childhood, but the remaining eight had always been in good health.

He had been temperate in the use of alcohol, but had smoked heavily. His surroundings had been comfortable, but his work had been hard; and he had been greatly exposed to the influences of the weather.

During the last five-and-twenty years he had met with several accidents. About twenty-four years ago he fell from the third floor of a house, and alighted on a stone stair upon the crown of his head. He was unconscious for about an hour, but was able to walk home afterwards, and observed no bad effects a few days later. At different periods subsequent to the date of this fall the patient had met with other accidents, some of which involved the head, but none of them seemed to merit special attention.

Nine months before coming to the Infirmary he observed a tendency to headache. At first it was slight and transitory, making its appearance towards evening, especially if the work of the day had been more arduous than usual; the pain, however, became more severe and persistent as time went on, and just before presenting himself the headache had been constant. Soon after the onset of the headache, the patient noticed some weakness of the left arm and hand, with difficulty in using the hand for complicated movements. Weakness of the left leg began to show itself at a later period, and walking became difficult in consequence. He subsequently observed occasional twitching of the left side of the face, accompanied by some interference with his utterance, which led some of his friends, at times, to express doubts as to his sobriety.

The patient was, on examination, seen to be a strong, muscular man, 5 feet 7 inches in height, and 10 stone 6 lbs. in weight, with a normal temperature. He complained of a feeling of coldness and numbness over the left forearm and hand. His perception of ordinary, painful, and thermal sensory stimuli was everywhere acute, quick, and accurate. The headache was referred somewhat indefinitely to the vertex, and on percussing that region a vaguely-defined area of tenderness could be determined. The pupils were equal, and reacted normally. The acuity of vision was unimpaired, and the field of vision unaltered. On examining the fundus of the eye with the ophthalmoscope, double optic neuritis was found to be present, the change being more advanced in the left eye. The patient could not hear so well with the left as with the right ear. The senses of taste and smell seemed unaffected.

Turning to the motor functions, it was found that the movements of all the muscles of the left side were much weakened. The left side of the face was deficient in expression, and the unilateral movements were distinctly diminished; he could, for example, only wink with the left eye in an imperfect manner, and his smile was mostly confined to the right side of the face. No involuntary twitching was observed at any time. The speech was somewhat indistinct. There was no deviation of the uvula or

deflection of the tongue on protrusion. The muscles of the left arm, and more especially of the left forearm, were a little smaller than those of the right, and they were deficient in tone. The power of the muscles on the affected side was considerably reduced, the patient being able only with difficulty to raise the arm to the level of his head, and, tested by the dynamometer, the grasp of the left hand was only 10, as against 55 on the right side. The loss of power was most marked, therefore, in the hand and forearm. The muscles of the left thigh were very slightly smaller than those of the right; the legs were equal in size; there was no loss of tone in the lower extremity. When walking, the patient slightly dragged the left foot. The left arm and leg, on being tested by electricity, showed a slight impairment of irritability to galvanic as well as faradic stimuli. The superficial reflexes were everywhere equal on both sides of the body. A well-marked wrist-jerk could be elicited on the left side, the left knee-jerk was exaggerated, and a slight ankle clonus was found on the same side. Muscular co-ordination was unaffected, and the muscular sense unaltered. The vasomotor and trophic functions were in no way impaired. The mental functions were in every respect good, except for a slight degree of irritability of temper, which was a new development in his disposition.

A careful examination of the head revealed a slight but distinct swelling situated  $3\frac{3}{4}$  inches behind the glabella, and nearly occupying the middle line, but lying more to the right side. The diameter of this swelling was about  $1\frac{1}{2}$  inches. It was hard and resistant, and was evidently of a bony nature. This area was the starting-point of the pain, and here the tenderness on percussion was best marked.

In this case no difficulty could arise with regard to the diagnosis. The general symptoms—headache, interference with speech, and optic neuritis—suggested the probability of a cerebral tumour; and the focal symptoms—shown by the distinctly localized motor disturbance—clearly pointed to the presence of a mass in the middle of the right motor area, occupying the centres for the left arm, and causing pressure upward upon the centres for the left leg, as well as downward upon those for the left side of the face.

As regards the nature of the tumour, it was not so easy to arrive at a definite opinion. As is well known, if syphilis be excluded, as it was in this case, about 80 per cent. of cerebral tumours are either tubercular or sarcomatous. Not a shred of evidence in favour of any tubercular tendency being present, we were inclined to the pathological diagnosis of a sarcomatous tumour.

The patient was placed under the care of my colleague, Sir Thomas Grainger Stewart, and entered Ward 22 on the day he was seen in the out-patient department. The nature of his case was fully explained to him, and, after consulting with Prof. Annandale, the advisability of seeking relief by means of an operation

was placed before him. As he would not entertain the proposal, he was treated by means of iodide of potassium and nerve sedatives.

The patient left the Ward on January 31, somewhat improved in regard to the headache, and feeling rather more power in his affected muscles. Although no longer resident in the Hospital, he attended daily as an out-patient, and was treated by means of strychnine hypodermically, with faradism to the paralyzed muscles.

The slight improvement which was manifested during the period of his stay in the Royal Infirmary was of brief duration, and the symptoms changed rapidly for the worse. The headache became so severe that sleep was greatly interfered with. The unilateral movements of the face and the movements of the forearm and hand on the affected side were completely paralyzed. The muscles of the shoulder and arm still retained some power of movement. The weakness of the lower limb had become so marked that the patient could not lift his foot clear of the ground. His memory was impaired, and his inhibitory power very markedly diminished. He was in consequence readmitted to the Ward, at that time under my care, on March 20, 1891.

The necessity of an operation was strongly urged upon the patient, and he gladly assented to the proposal. He was therefore transferred to the care of Prof. Annandale on March 24.

The following day Mr Annandale operated. The bone was removed from the region corresponding to the middle third of the motor area on the right side. Both bone and dura mater seemed quite healthy, but on cutting through the latter a soft reddish mass was exposed, which was easily separated by the finger from the surrounding brain tissue. As the mass was too large to be removed in one piece without greatly enlarging the opening in the skull, Mr Annandale divided it, and extracted it in several portions. The tumour was examined by Dr Barrett, who found it to be a glio-sarcoma.

After the operation the patient made an uninterrupted recovery, and has remained in good health ever since. He has recovered the use of almost all his muscles, he can walk a dozen miles, and has nearly equal power in both arms. With the dynamometer the grasp shows 80 with the right and 70 with the left hand. The more specialised movements, however, of the left hand have not been restored, and there is still an exaggerated jerk at the left elbow and wrist, and an increase in the left knee-jerk, along with ankle clonus; there must, therefore, be permanent degeneration of the motor tracts descending from the right side of the brain. It is a most interesting fact that Dr Argyll Robertson and Dr Mackay could trace no vestige of optic neuritis in either eye when he was seen by them in July 1895.

The great lesson which has been borne in upon me by this case, which has already been fully recorded by me,<sup>1</sup> is the vital import-

<sup>1</sup> *International Clinics*, vol. ii. (second series), p. 131, 1892.



ance of early operation. Had the patient yielded to our advice, and allowed us at once to give him the opportunity of surgical relief, it is very probable that he would have regained more of the power of finer adjustment in the hand of the left side.

## II. CEREBRAL TUMOUR PRODUCING COMA.

*(Reported by Dr J. G. Cattanach.)*

R. G., 50, formerly soldier, afterwards a house-painter, and latterly a park-ranger, was admitted to Ward 22 under my care on 13th April 1893, on the recommendation of Dr White. The patient had been for twenty-one years in the army and had suffered from syphilis. His second wife had borne two still-born children. Five weeks before admission his lips began to be drawn to the right side. A week later he complained of headache, at first frontal, later vertical, but towards the right side, and over the right side there was tenderness. He also began to suffer from spasms of the great toe of the left foot. He was treated with iodide of potassium, which relieved the headache, but four weeks before coming in he began to drag the left leg, which felt numb, and he could not hold objects in his left hand, but let them fall.

On examination the patient was found to be torpid, with great tendency to stupor; there was restlessness and confusion of ideas. There was some cyanosis of the lips. The tongue was furred and tremulous. The temperature was subnormal, pulse about 80, breathing 14. From the mental condition of the patient it was impossible to determine the state of the sensory functions. The pupil reflex to light was very sluggish. Intense double optic neuritis was present. Swallowing was imperfectly performed. Micturition was interfered with, retention and incontinence being occasionally noted.

On getting the patient out of bed it was seen that the left leg could not be raised at all, and an attempt, when supported, to walk, showed that the affected leg was simply dragged along. The left arm was almost quite paralyzed as well as the left side of the face. The left knee-jerk was much exaggerated, and there was left-sided ankle clonus. The patient understood what was said to him, but was scarcely able to reply, and any words employed were pronounced in a slow and slurring fashion. He was treated energetically with iodide of potassium.

During the next four days the tendency to stupor became more marked, so that by the 17th April the patient was quite comatose. The left leg and arm became absolutely powerless, but the face showed distinct twitchings on the left side.

After consultation with Mr Alexis Thomson it was decided that an operation was not only justifiable but necessary, and the patient was transferred to Ward 7 on the 18th April. As the symptoms pointed to an implication of the entire motor area on

the right side of the brain, the site selected for operation was the middle zone of the Rolandic region. On removing the bone the dura mater was seen to bulge very markedly, and on reflecting the membrane the exposed brain swelled out and showed no pulsation. The surface of the brain seemed normal in colour and consistence. On incising the brain substance and introducing the finger nothing abnormal could be felt. The lower zone of the Rolandic area was then exposed, also with negative results.

On the following day the patient was somewhat improved. He expressed himself freely and sensibly in answer to questions, and said he felt much better. He put out his tongue readily, and took food with relish. He had, however, no return of power in the muscles of the left side. Two days later he became delirious, and muttered quietly. He had some ptosis of the left eye, and internal squint of the right. Involuntary micturition was present. On the 22nd April he again became sensible, recognised and conversed with his wife, but in the afternoon he collapsed suddenly, lay for a time in a state of coma, with the eyes turned to the right, and died quietly.

The post-mortem examination, performed by Dr Leith, showed that there was no sepsis, and that the wound had almost entirely healed. No meningitis was present. On slicing the brain, a tumour, of a greyish-red colour, was found in the right hemisphere. It was about 1 inch in transverse and  $\frac{3}{4}$  inch in antero-posterior measurement, and occupied the corpus striatum. The mass was outside of the caudate nucleus, and occupied the knee and the anterior portion of the posterior limb of the internal capsule, encroaching on the lenticular nucleus, and pressing upon the corpus callosum. In the posterior part of the brain there was some encephalitis. The microscopic investigation of the tumour showed it to be gliomatous in structure.

From this case, which had, unknown to me, been seen previously by Dr Bramwell, and to which he referred in a recent communication,<sup>1</sup> some useful deductions can be drawn. In the first place it is evident that even Jacksonian spasms may have little localising value. Judging by the symptoms, a lesion might reasonably have been expected in the cortex, beginning in the centre for the lower limb and invading the centres for the arm and face. The case further shows that on the removal of the increased intracranial pressure consciousness was for a time restored.

It may be asked whether, in a case like this, if the diagnosis had been correctly made of a tumour in the region of the great basal ganglia, operation would have been justifiable. To my mind it would be not only justifiable, but even necessary. Sir Thomas Watson in his classical lectures recommends, in the case of patients dying of cholera, the transfusion of warm water into the veins with

<sup>1</sup> *Transactions of the Medico-Chirurgical Society of Edinburgh*, vol. xiii., new series, p. 132, 1894.

the object of allowing the opportunity of making a will.<sup>1</sup> How much more urgently is surgical interference demanded in such a case as this, with the possibility of finding a lesion, supposed to be inaccessible, situated in such a position as to allow of its removal!

### III. CEREBELLAR TUMOUR.

(*Reported by Dr Purves Stewart.*)

I. K., a married woman, æt. 25, was admitted on 8th April 1895 to Ward 25 of the Royal Infirmary, then under my charge, on the recommendation of Dr Laing of Dundee. She complained of headache, giddiness, and difficulty in walking. These symptoms had commenced ten months before admission, and had steadily grown worse in spite of treatment.

Her family history was satisfactory, there being no evidence either of tubercular or of inherited specific disease. She had been married for three and a half years, and had borne one child, who died from convulsions at the age of eight months, four weeks before patient's admission to Hospital. When three months old the child had on its face and body a rash, the exact nature of which the mother could not describe. The patient never had any miscarriages. There was no history of syphilis, either primary or secondary. The health of her husband, who is a sailor, could not be ascertained.

The patient was examined on the day after admission, and the following conditions were found in the nervous system:—As regards the sensory functions, there were no subjective sensations of any sort, with the exception of a deep-seated headache, which patient described as feeling "like a sharp dagger," strictly localised to the frontal region, exactly over the left eye. This pain was not increased by firm pressure, nor by tapping over the painful area. There was no cutaneous anaesthesia anywhere, and the muscular sense was normal. On examination of the eyes there was found constant marked nystagmus, both in the horizontal and in the vertical meridians. The left pupil was larger than the right; both pupils reacted to light and to accommodation. On ophthalmoscopic examination distinct optic neuritis was found in both eyes. Although the external and middle ears were normal, yet patient was completely deaf in the right ear, both to external sounds, such as the ticking of a watch, and to a tuning-fork placed on the vertex. Taste and smell were normal.

As regards the motor functions, there was no motor paralysis or paresis, with the exception of the soft palate, the functions of which seemed to be slightly impaired, as evidenced by the regurgitation of fluids through the nose during swallowing. Her voice

<sup>1</sup> *Lectures on the Principles and Practice of Physic*, fourth edition, vol. ii., p. 528, 1857.

was somewhat "bleating" in character, and she herself noticed that it had altered from its normal state. The larynx was not examined. The organic reflexes were normal, with the exception of deglutition, which was occasionally a little difficult, especially on attempting to drink large draughts of fluid. The skin reflexes were normal. Both knee-jerks were equally exaggerated, and occasional ankle clonus could be elicited on both sides. The patient's gait was very pitching and staggering in character: she reeled along, walking on a broad base, planting her feet widely apart, but neither stamping her heels nor scraping her toes. She complained of distressing vertigo, but only when she moved in bed, or tried to sit up or walk. So long as she lay still there was no feeling of giddiness. Her gait was so unsteady that she inclined to fall unless supported, and there seemed to be a tendency to fall on the right side. On making patient stand, with her eyes shut and feet together, the swaying was very marked, and here also she tended to fall to the right side.

There were no vasomotor or trophic changes. The patient's intelligence, attention, and memory were excellent. She slept badly, owing to the persistent headache, which was always worst at night. There was no abnormality to be discovered on examination of the cranium and spine. As regards the other systems, the alimentary system was normal, save for a slight tendency to constipation; the lungs and heart were quite sound. As to the integumentary system, there was a small patch of ichthyosis over each ligamentum patellæ. With respect to the reproductive system, patient had amenorrhœa of four months' standing. The urine contained no abnormal constituents. Prior to admission, the patient had not suffered from vomiting, but this symptom supervened a few days after she came into Hospital. This vomiting recurred at intervals, at first of several days, but later more frequently, and it was quite unassociated with the ingestion of food.

Such being the clinical facts, the question of diagnosis next arose. The presence of headache, vomiting, and double optic neuritis indicated some intra-cranial lesion. The absence of distinct motor paralysis, either of limbs, trunk, or face, combined with the presence of a staggering gait and distressing vertigo, pointed clearly to the cerebellum as the seat of the lesion.

On attempting to attain to a more definite localisation, there were several points to guide us. The tendency to fall towards the right side is usually regarded as significant of right-sided cerebellar lesions, and had some weight in considering the case. The right-sided labyrinthine deafness showed that there was some lesion of the auditory path on that side. According to Meynert, after leaving the nucleus the auditory tract passes through the cerebellum, and whether this be allowed or not, there can be no doubt that the right-sided deafness points to the probability of

pressure upon some part of the auditory path or its nucleus. It seemed clear that the affection was right-sided, and on account of the trouble connected with deglutition as well as with hearing it seemed probable that it was situated at a low level.

But in addition to these facts there was another point which was of much interest. It is one which has not, so far as is known to me, been previously described as of diagnostic import, and although we cannot as yet assign any definite position to it, it appears to me to have the promise of the highest value. Dr Purves Stewart, resident physician at the time, called my attention to the persistent headache in the left frontal region, and suggested that it might be produced by irritation of the fronto-cerebellar fibres crossing from the left frontal region to the superior cerebellar peduncle. It affords me much pleasure to have this opportunity of stating that, in my opinion, he has added a valuable symptom to the diagnostic means at our disposal in studying cerebellar disease. Whether it may ultimately prove of as much use as we hope cannot at present be foreseen, but some corroborative facts have already been brought under my notice which induce me to cherish the expectation that this new symptom is one holding out the promise of real utility.

From a consideration of these facts, the diagnosis was made that there was a tumour in the lower part of the right lateral lobe of the cerebellum. Whether the tumour was of the nature of a gumma, a tubercular mass, or a new formation, could not be determined. With the view of eliminating as far as possible the chance of the lesion being a syphilitic one, the patient was at once placed under treatment, by gradually increasing doses of iodide of potassium, until on 20th April she was taking 60 grs. three times a day. In addition to these massive doses of iodide, inunction with blue ointment was applied every day to two parts of the head,—over the right half of the cerebellum, and over the seat of pain in the frontal region. This treatment was continued until 30th April. So far from improving, the patient grew worse; all her symptoms became more aggravated, and more especially the vomiting became more frequent. On 30th April, therefore, after consultation with Professor Annandale, she was transferred to his Ward, with a view to surgical interference. After her admission the vomiting increased still more in frequency, and the patient was evidently going from bad to worse.

On 3rd May Professor Annandale cut down over the right lobe of the cerebellum, trephined the skull, and discovered a tumour about half an inch below the surface. The tumour was about the size of a pigeon's egg, and lay low down in the lateral lobe of the cerebellum, rather closer to the foramen magnum than to the outer wall of the skull. On opening into the mass, about a drachm of clear serous fluid escaped and was lost. Within the cyst was found a solid tumour, apparently encapsulated. This was completely

removed piecemeal, and subsequent microscopic investigation showed it to be of the nature of a fibro-sarcoma.

The progress of the case after the operation was eminently satisfactory. When the patient recovered from the chloroform anæsthesia the frontal headache had entirely disappeared, and has never returned again. The sickness also entirely ceased, the nystagmus became much slower and less marked. Instead of being constant, as it was before the operation, it occurred only when the patient fixed her eyes on some object not directly in front of her. When the globes of the eyes were in a position of rest—that is, looking straight forward—there was almost no nystagmus. The feeling of giddiness on changing her position also entirely disappeared. Her swallowing became quite perfect, even with fluids. Her gait much improved. The optic neuritis almost disappeared.

Since leaving the Royal Infirmary the progress of the case has been most satisfactory, as may be seen from the letter now subjoined:—

“9 Tay Square, Dundee, Nov. 18, 1895.

“MY DEAR DR GIBSON,—I have just received your kind note about Mrs K. I delivered her of a healthy girl about three weeks ago, after a perfectly easy and normal labour. She made an excellent recovery, and is now very well. Had it not been for this she would have been across to report herself, and no doubt she will do so when she can. She walks quite well now, speaks as she used to, and has no headache or giddiness. There is still a little nystagmus, and over the site of the operation a painless fluctuating swelling still remains. In all respects it has been a most satisfactory result.—With kind regards, believe me, very sincerely yours,

“JAMES H. W. LAING.”

But little commentary is necessary on this case, the full details of which have been placed on record by Dr Purves Stewart,<sup>1</sup> but it now seems to me quite clear that it would have been better for the patient if we had operated earlier, as we might then have got rid even of the slight nystagmus still present.

#### IV. INFANTILE HEMIPLEGIA.

(Reported by Dr A. M. Easterbrook.)

W. M., schoolboy, aged 13, was brought on several occasions to see me in the waiting-room, complaining of paralysis of the left side of the body, especially of the left leg, and fits. The patient's father is alive, but subject to bronchitis; his mother enjoys good health. There seems to be, however, a bad history of parturition on the part of the mother. The patient had four brothers, all dead; two only survived a few hours after birth, and one about a day and a half after birth; the remaining brother died of consumption. The patient says one cousin has paralysis of the right side, but he thinks it was the result of an accident. The patient says he has a

<sup>1</sup> *Edinburgh Hospital Reports*, vol. iii. p. 447, 1895.

pleasant and comfortable home, and that the locality, as far as he knows, is healthy; the drains seemed to be out of order, however, when he was attacked by diphtheria. He has not had the usual child's complaints (as measles, scarlatina, whooping-cough); he has had bronchitis several times. About two years ago he was in a private hospital with diphtheria for seven weeks; he does not seem to have suffered from post-diphtheritic paralysis, nor was his left-sided paresis aggravated by the diphtheria; he has had no other serious illness except the present condition. The patient says he has had the paralysis ever since birth, and he says his mother ascribes his condition to an injury he received at birth on the right side of the head; the labour was evidently a difficult one, and forceps were used. His mother told him that the doctor used to come and manipulate his head for some days after the birth, and expressed hopes that the child would recover from the injury, but the whole of the left side of the patient's body has ever since birth been paretic and weaker than the right side, and has had a tendency to be ill-developed; he has evidently had talipes equinus of his left foot since his childhood, as he says he always walked as he does now. He has been treated for his condition, but has not made any marked improvement, although he says that now he has more power in and can use his left arm better than before. With regard to the fits, he states that he commenced to have them about twelve months ago, and at first he used to have two, three, and four in a week, but of late (for the last month or two) they have diminished in frequency, he only having now about one a week. He says he can usually tell when he is going to have a fit, although he cannot describe any very definite aura immediately preceding the fits, but he usually has a headache for about a day before, and sometimes feels sick for some hours before a fit; and when actually going into the fit he says he usually becomes giddy, and everything seems to whirl round about him. He says he does not always lose consciousness during a fit, but often does so.

On admission the patient was seen to be fairly well nourished, but the left side of body was not so well developed as right. The patient's left upper extremity seems to be slightly less developed than the right, and there seems to be some degree of permanent contracture of the left biceps, causing the left elbow to assume the flexed position. The left lower extremity seems slightly less developed than the right, and there is marked talipes equinus of the left foot, and also some degree of inversion of the left foot. The patient looks anæmic, and his head is inclined to droop forwards on the chest. He has rather prominent eyeballs, and at times a somewhat vacant stare. His face generally is somewhat expressionless; his temperament seems to be rather of a nervous character, and he has not a very intelligent expression. When spoken to, however, he seems to have a fairly good memory and intelligence, but is apt at times to be slightly dazed and vacant.

This is not, however, by any means a marked feature. His muscles, generally speaking, seem to be subject to small jerky, spasmodic movements, which seem to be involuntary. There are no marked fibrillary twitchings. The patient is subject to pain in the head, especially on the right side and in the frontal region. When it is present, he says it is a dull aching pain, and that it makes him feel dizzy and unable to do anything, and he has to go to bed whenever it comes on; the patient says he always has this headache before and more severely after a fit. His headache, however, is sometimes present without a fit necessarily occurring. He also experiences sometimes a stiffness and pain round the neck, especially at the back of the neck,—it prevents him from moving his neck freely. The patient also sometimes experiences sensation of pins and needles down the whole left side of body, especially in the left foot, not much in the left arm. The sensation of pins and needles is present sometimes apart from fits. He also says that his whole left side feels asleep when he is going into a fit, and for an hour or so after he has come out of a fit. He also experiences tingling sometimes in the left upper and lower extremities. No history of lancinating or girdle pains, with the exception, perhaps, of that occasional stiffness he experiences in the neck. The tactile sensibility seems to be unimpaired; he can always tell you where you are touching him when his eyes are closed. Perhaps at times he is rather slow in localising the spot, but on the whole his sensation of touch is fairly normal. Sensibility to heat and pain are all unimpaired. After examining him for some time he has made mistakes with regard to heat sensation on his left leg, but he is nearly always right in distinguishing between hot and cold test-tubes. The muscular sense seems to be unimpaired. He can balance himself quite well with his eyes shut, but has some difficulty in so doing, even with his eyes open, when he puts both feet closely together, due to talipes, perhaps. The special senses are unimpaired. The organic reflexes are normal, except that the breathing tends to be jerky sometimes, and he says he is breathless when coming out of a fit; also the heart's action is irregular. The superficial reflexes are little affected. Ankle clonus is markedly present on the left side; it can occasionally be produced on the right side. The knee-jerk is distinctly more marked on the left side; and knee clonus can also be produced on the left side by repeated tapping over the quadriceps extensor tendon. The wrist-jerk is well marked on the left side; not so on the right side. The patient's voluntary motor power is somewhat impaired on the left side. This is most marked in comparing the two upper extremities, the left arm being distinctly weaker than the right arm. No special groups of muscles seem to be involved. The power of co-ordination is not very great in either upper or lower extremities, but better on the right side than on the left. He cannot describe a circle well with either



foot, but a better attempt is made with the right foot. He cannot walk along a straight line very well; this is perhaps due to a certain extent to his deformity on the left side,—talipes equinus. He can pick up objects well enough, but is apt to be slow in his movements. On being asked to touch various parts of his body with his eyes shut, he goes wider of the mark with his left hand than with his right. As a rule, he puts his finger within two or three inches of the spot he is asked to touch. The skin is dry and fairly well nourished on both sides. The soft tissues of the left extremities are somewhat wasted,—*i.e.*, not so well developed as on the right side. The left lower and upper extremities tend to be colder than those on right side. There does not seem to be any tendency to glossiness of the skin on the left side or elsewhere. There are no eruptions except a papule on the nose, and the mark of a dog-bite on the back of the right popliteal space. The intelligence is fairly good. He is apt to be distracted and dazed at times (especially after much examination). His memory is not particularly good. His speech seems to be normal, but patient cannot read or write well—only very simple words, as dog and cat. He can only write simple words and his own name, and only in badly-formed capitals (probably due to neglect of education). The patient sleeps well, as a rule, except when his headaches come on, and these then prevent him from sleeping. He does not seem to be troubled with dreams. There is a general fulness to be made out over the right temporo-parietal region. No special prominence or thickening of bone is to be made out at any one point. No scar is observed on head. There is no specially painful spot to be made out by percussing all over the head. On inspection, a slight left lateral curve is seen to be present in the dorsal region. The first three lumbar spines are perhaps unduly prominent. Percussion down spine and hot sponge test elicit nothing of importance. The result of careful measurement of the bones and muscles shows no striking differences between the two sides.

The patient was brought to see me at the waiting-room on several occasions, and was treated by means of tonic remedies along with the bromides. As he made no progress in any way he was taken into Ward 22, then under my care, on 18th April 1894. It seemed to me that there were two possibilities,—he might be afflicted with hemiplegia dependent on porencephaly, or caused by some localised lesion on or near the motor area. On consultation with Mr Alexis Thomson, we agreed that in view of the cranial accident at birth there was at least a possibility of the latter alternative, and resolved to operate, a course that was eagerly sanctioned by the parents. The patient was therefore transferred to the surgical side.

Mr Thomson removed a large piece of the skull in the situation of the right motor area, but failed to see any alteration in the membranes or cortex. He therefore sewed the

membranes, replaced the bone, and closed the wound. The boy made a perfect recovery from the operation, but except for some increase of intelligence has not in any way improved. We were not, in this instance, sanguine as to the possibilities open to us; but after reviewing all the facts and circumstances presented by the case, it seems to me that we should with similar conditions follow the same course as on this occasion.

#### V. GENERAL PARALYSIS WITH EPILEPTIFORM ATTACKS.

*(Reported by Dr J. G. Cattanaeh.)*

J. R., aged 47, domestic servant, was admitted to Ward 6 on 4th December 1894, on the recommendation of Dr Crawford Dunlop.

About twelve years before admission she was struck on the head by a burglar. She was for some time unconscious, and was never able to give any clear account of the assault. Four years before admission she began to take fits, and had since then suffered from convulsive attacks at irregular intervals. These were said to have commenced invariably by spasms of the right arm, spreading over the entire body. Latterly after the attacks she was in a more lethargic condition than used to be the case. In September the patient was placed in the Elgin Asylum on account of her mental condition. While there she had two convulsive attacks, and more than one maniacal outbreak. The convulsions were said to have begun in the right arm, and to have been almost confined to that limb.

On examination the patient presented no change in sensibility, common or special. There was no optic neuritis. The organic and superficial reflexes were unaltered, and there was no change in the myotatic irritability. The intelligence was weak. Questions received irrelevant replies, and the patient was moved to tears and laughter by slight causes. No convulsive attack took place while the patient was under my care, although she was watched day and night. The relatives of the patient were very anxious that some attempt should be made to ascertain the cause of the convulsive attacks, and to remove it if possible. Mr Cotterill accordingly saw her, along with Dr Dunlop and myself. It seemed to us that the patient suffered from dementia, and that it was possible there might be some focus of irritation on the right side of the cortex. It was therefore determined to have recourse to operation.

On 20th December Mr Cotterill, with the wheel-saw, turned back a flap of bone about the size of the palm of the hand and exposed the motor area on the left side. There was no thickening of the bone or dura mater, and on reflecting the latter no increase of fluid could be seen. The surface of the brain, however, was somewhat flattened, and showed an opalescence rather suggestive of some old-standing hyperæmia. On stimulating the motor area with a weak faradic current there was a sluggish reaction of the leg and arm,

but the side of the face was thrown into typical epileptiform spasms. By means of a fine trocar and cannula a small quantity of fluid was drawn from the lateral ventricle, the dura mater was stitched, the bone replaced, and the scalp sutured.

The patient spent a very restless, noisy night. The temperature rose to 100° and the pulse to 120, but the respirations remained normal, about 17 or 18. On the following day she was still very restless; the pulse, temperature, and respiration rose. During the next two days the symptoms became more serious, and early in the morning of the 24th December, after the temperature had risen to 102°·6, the pulse to 162, and the breathing to 52, she passed quietly away.

The brain was forwarded to Dr W. F. Robertson, pathologist to the Royal Asylum, who kindly furnished me with the following report:—

“Royal Asylum, Morningside, 25th December 1894.

“DEAR DR GIBSON,—Many thanks for the interesting brain you sent me yesterday. I have cut and examined sections, and now send you one. Histologically the case is one of general paralysis of the insane, and I have little hesitation in saying that the case was really one of that kind. Dr Cattnach has sent me a note of the history, which is quite consistent with this view, though somewhat exceptional. You will notice marked spindle-cell hypertrophy, not only in the first layer, but in the depth of the cortex and in the white matter; thickening of the capillaries, aggregation of round cells upon the larger vessels, and advanced degeneration of the nerve cells. The last is chiefly pigmentary, but there is also a marked degree of nuclear vacuolation. This, as you no doubt remember, Dr Bevan Lewis specially associates with epilepsy—an observation which we have not been able to corroborate at Morningside. I had quite recently a case of general paralysis which showed as marked nuclear vacuolation as is seen in this case.—Yours very sincerely,  
“W. F. ROBERTSON.”

Now, in this case there was an error in diagnosis; the case seemed one of dementia, with a possible focus of irritation. If it had been regarded by me as one of general paralysis, looking to the published results of operative measures in that disease, it would have appeared undesirable to have adopted any surgical procedure. But, considering the condition as one of dementia, and with the history of Jacksonian spasms, there seemed to be a very clear duty in the case. Dr Clouston, in a private communication, informs me that alienists make about two per cent. of errors in the diagnosis of general paralysis, and he is kind enough to indicate that he would allow the ordinary physician a wider margin!

## VI. OLD FRACTURE OF VERTEX OF SKULL WITH SEVERE PAIN.

(Reported by Dr Purves Stewart.)

C. M., 55, rivetter, presented himself before me as an out-patient, and was admitted on 16th April 1895 to Ward 22 under

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my care. Thirty-three months before, he had been injured by a plank which fell 30 feet, and struck him on the head, fracturing at the same time his left clavicle and left radius. For two days he was unconscious, and was under treatment in Sunderland Infirmary for two months. During the interval which had elapsed since then he had for the most part been in bed suffering from occipital headache, giddiness, inability to walk, and deafness of the left ear, attended by blowing noises. He had never been subject to any spasmodic symptoms throughout. The patient was well nourished; his expression was vacant, his apprehension dull, and his speech sluggish. On the left side of the scalp there was a curved scar  $2\frac{3}{4}$  in. long near sagittal suture, with convexity towards it; the posterior end of the scar was  $6\frac{3}{4}$  in. from the glabella, and  $1\frac{1}{4}$  in. from the middle line, and its direction was towards the temple. The bone beneath showed a depression. There was no tenderness; none of the muscles were in a state of paresis; the patient could not turn quickly or walk long; the left knee-jerk was about normal, the right diminished; there was no clonus; both eyes had slight hyperæmia of nasal part of the disc.

On consultation with Mr Alexis Thomson it was resolved that, as the symptoms were undoubtedly due to the injury, exploration of the site of the lesion would probably afford relief, although it might fail to disclose anything more than thickened bone, and the patient was therefore transferred to Ward 7, under Mr Thomson's care, on 22nd April.

On the 23rd April Mr Thomson reflected a large scalp flap, and with the wheel-saw removed an elongated segment of bone, including the area of depression. The inner table appeared smooth; there was considerable bleeding from the diploë, the dura mater was much thickened, and on opening it more than the usual amount of cerebro-spinal fluid escaped. The brain surface appeared healthy. After stitching the dura mater, the bone was replaced and the flap sutured.

The patient recovered rapidly from the effects of the operation, and left the Infirmary quite free of pain,—showing, however, to the time of his departure the slight change in the optic discs. Commentary on this case is needless.

## VII. FRACTURE OF SKULL, WITH COMPRESSION OF BRAIN FROM HÆMORRHAGE.

*(Reported by Dr J. G. Cattanach.)*

J. R., 50, labourer, was admitted to Ward 6, under my care, 6th January 1895. For some weeks he had been drinking heavily. On the morning of the 5th January he had a fit in his lodging, and afterwards went out. He was picked up in an unconscious state in the street by the police, and detained

over-night as being drunk and incapable; but as he was still unconscious on the following morning, he was brought to the Infirmary.

He was, when examined on 7th January, found in a comatose condition, with a temperature of  $103^{\circ}$ , a pulse of 136, and breathing of 42. The breathing was stertorous, the pupils unequal, the right being contracted, the left dilated. There was a cut over the left parietal eminence. Twitchings were seen on the right side of the face.

On the 8th January the patient had a localised convulsion, commencing with a spasm of the muscles of the right side of the face, and spreading to the arm and leg of the same side. This hemispasm was repeated about every ten minutes. In the intervals between the attacks the muscles of the affected side were limp and flaccid.

On consultation with Mr Alexis Thomson, it was resolved that, as the patient was apparently dying from compression primarily of the left cerebral cortex in the motor area, it was a duty to give him the possibility of operative relief, and he was accordingly transferred to his care.

Mr Thomson raised a flap over the left arm and leg areas, and with a wheel-saw removed a piece of bone. The dura mater was yellowish in patches, bulged considerably, and did not pulsate; when incised an extensive thick blood-clot was found. This was as far as possible washed away, the dura mater was sewn, the bone was not replaced, and the flap was sutured.

The patient was decidedly more sensitive to stimuli after the operation, and remained free of spasms for about two hours, but after that interval he began to have twitchings of the right side of the face, which recurred about three times an hour, but finally ceased nine hours after the operation. He had involuntary micturition, but looked about him. At 10 o'clock on the morning of 9th January he died quietly.

At the post-mortem examination by Dr Muir, on 10th January, a fissured fracture was found, extending from the left parietal eminence to the middle fossa of the base. A considerable amount of blood had escaped, and there was laceration of the right temporal lobe of the brain by *contre-coup*. All the arteries were atheromatous, but it was impossible to detect the source of the hæmorrhage. Any remarks upon this case would be superfluous.

The principal conclusions to which the consideration of these cases has led me have been stated in connexion with the narration of each. It only remains to sum them up—an easy task, as they are almost entirely comprised in the advice to operate early, which can only be rendered possible by the loyal co-operation of the physician and the surgeon. We sometimes hear the statement

that the diagnosis of such cases belongs solely to the physician, and that when he has made up his mind he has simply to issue instructions to the surgeon. In fact, we are often told that the surgeon is simply the hand, the physician the head. This, however, is a point of view that should be warmly repudiated. Not only does it throw discredit upon a great branch of our profession, but it also renders the mutual helpfulness of medicine and surgery impracticable, and prevents the full benefits which accrue from the harmonious co-operation of real fellow-workers.

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*The President* said they had had a very interesting series of cases of cerebral surgery brought before them, and not the less interesting because it had been brought before them by a physician. These cases were interesting, not only to the physician and surgeon, but to the neurologist, oculist, and dentist—in fact, to all practitioners of medicine. He had no doubt they had been listened to with much interest. The hour was late, and he hoped that those who spoke would be as brief as possible.

*Mr Caird* said that this interesting series of cases strongly emphasized the position that every physician should take up, viz., that he should recognise how much aid he could obtain from the surgeon, especially if he were called early. One question of great interest was—how long might optic neuritis exist, and to what extent, and yet after removal of intra-cranial pressure by trephining return to the normal?

*The President* said he had seen several cases of very acute, thoroughly developed optic neuritis in patients suffering either from cerebral tumours or injuries, in whom operations had been performed relieving intra-cranial pressure, and with the result that very good, he might say perfect, vision had been restored in some instances. Whether perfect vision was restored or not depended on a variety of circumstances. One most particularly was the length of time it had existed; whether the inflammatory changes had gone on to such an extent as to induce atrophy of the optic nerve fibres. From that condition recovery could not be expected, but anything short of atrophic conditions might be recovered from in these cases. It was well known that there might be extensive optic neuritis with scarcely any perceptible diminution in vision, which arose from the circumstance that the impression which was found on the retina was accurately perceived by the retinal elements, and was conveyed to the brain, and there accurately perceived by the patient. This condition of affairs remained perfect so long as atrophic changes did not occur in the optic nerve. That was the condition on which the final result of the case depended. If the optic changes had advanced to a considerable extent, then only imperfect restoration of vision could be expected. But he could not give any definite statement

as to when the atrophic changes occurred. That depended on the extent of the optic neuritis. Some cases of comparatively mild optic neuritis might run a prolonged course before giving rise to atrophic change, while others of a more acute severe type very soon produced the changes.

*Dr Purves Stewart* said he would like to call attention to the new symptom localising cerebellar lesions. All the localising symptoms had been hitherto associated only with the middle lobe of the cerebellum, which was its essential part. As they passed down the animal scale the lateral lobes became smaller, until in birds they disappeared. They would expect then most of the localising symptoms in affections of the middle lobe; but in the case to which Dr Gibson had referred, the tumour was successfully diagnosed in the lateral lobe, and that by means of a new symptom, viz., the fronto-cerebellar headache. The tumour was on the right side, and the headache in the left frontal region; and the reasons for giving the diagnosis were chiefly anatomical. The cerebellum had three sets of peduncles—inferior, middle, and superior. The inferior peduncles or restiform bodies connected the upper part of the middle lobe of the cerebellum with the nuclei of the posterior columns of the cord. Some of its fibres decussated as they passed downwards, others remained uncrossed. The middle peduncles of the cerebellum formed the transverse fibres of the pons, which were formerly considered to be mainly commissural, but more recently it had been shown that this was not so, but that the transverse fibres of the pons arose mostly in the cortex of the cerebellar hemispheres, and passed across the middle line to nerve cells in the pons, which cells, again, were connected with the various lobes of the cerebrum. Each superior cerebellar peduncle arose mainly in the interior of one dentate nucleus, but also in part from the cortex of the cerebellum, and especially from the lower part of the lateral lobe. Its fibres, when traced upwards, were found to decussate with those of the opposite side, between the corpora quadrigemina. Some of the fibres appeared to end in the red nucleus of the tegmentum of the opposite side, whilst other fibres passed this nucleus, and ran on to the frontal lobe of the opposite cerebral hemisphere, thus constituting a fronto-cerebellar tract. The question came up, which part of that long tract were they to fix the lesion in? The right labyrinthine deafness did not help them much, because there were two views as to the auditory path. One view was that it ran up in the tegmentum; others, on the contrary, *e.g.* Gowers, held that it ran through the cerebellum itself; but the patient had some paresis of the palate, some difficulty of swallowing, and some weakness of the vocal cords, which indicated pressure on the bulbar part of the spinal accessory, and that the lesion therefore was compressing the medulla, and that if it were in the superior peduncle it must be low in it. Mr Annandale cut down on the

lower part, and removed a tumour of the size of a hen's egg from a position midway between the medulla and the side of the skull. Since the publication of this case he had obtained notes of another case with a similar symptom, but in which the diagnosis was not made, viz., that of a woman of about 50, with headache, vomiting, double optic neuritis, giddiness, exaggerated knee-jerks on both sides, and history of convulsions not accompanied by unconsciousness, beginning in right arm,—the patient going from bad to worse in spite of potassium iodide. The surgeon trephined over the left frontal region, and found nothing abnormal. The patient died two days afterwards, and post-mortem the tumour was found in the right lateral lobe of the cerebellum. He (Dr Stewart) had been in communication with Dr Risien Russell, and also with Dr Gowers, and they had been good enough to admit the feasibility of the hypothesis which had been advanced.

*Dr William Russell* said he would like from the physician's side to express his very great admiration of the series of cases Dr Gibson had laid before them. These cases did not lend themselves to criticism, but were valuable as being one man's entire record—some eminently successful, others moderately, others not at all. That was the kind of record that they wanted there, and as a profession, both with reference to cerebral surgery and other things, and they did not always get it. He thought they might congratulate Dr Purves Stewart on having so early in his career discovered a symptom which, he was sure, they all hoped would be found reliable and useful in the diagnosis of those important cerebellar lesions.

*Dr Gibson*, in reply, said that there was very little for him to say, because the Society, with its usual generosity, had accepted the observations brought before them with characteristic kindness. He had to thank the President for his observations on optic neuritis, which practically coincided with what he had in a much smaller way always seen; and he desired also to say how much he appreciated the remarks made by Dr Russell. He was one of the most ardent advocates of Dr Purves Stewart's views in regard to the anatomical aspect of the case to which he had specially referred, but he was of opinion that the diagnosis was founded on a sure basis, even if the symptom now for the first time described had been absent. It was, however, with much interest that he had followed Dr Purves Stewart in working out the details connected with the anatomical investigation of the symptom in question, and he felt sincere pleasure in having this occasion of congratulating him on the discovery of what promised to be a valuable aid to diagnosis.



## Meeting III.—December 18, 1895.

Dr WILLIAM CRAIG, *Vice-President, in the Chair.*

## I. EXHIBITION OF PATIENTS.

1. *Dr Allan Jamieson* showed a case of XERODERMA PIGMENTOSUM IN AN EARLY STAGE. Janet W.,  $3\frac{1}{4}$  years old, was brought to the Royal Infirmary on 16th November 1895. She lived in the outskirts of Edinburgh, and was an only child. Her mother's father died of cancer of the tongue at the age of 69, but he had been a very heavy smoker. Several near relatives on both sides have died of consumption. She was born at seven months, yet she thrived well till she was a year old. Vinolia soap was used to wash the child. When twelve months old her mother noticed a roughness of the face, such as might be caused by cold. The skin was itchy, for the infant scratched it. This roughness gradually became worse, though always confined to the face. The surface never oozed or became wet; was always dry and scaly. There were then no freckles. These appeared nine months later, and could not be accounted for by any undue exposure to the sun's rays. Since then they have increased in number. The child is well enough grown for her age, is nervous, fretful, and capricious. On admission it was seen that only the skin of the face was affected; there were no freckles or any abnormal changes on the neck or hands. The skin of the face was pigmented as well as being much freckled. It was dry, rough, and very slightly scaly. Here and there were stigmata or telangiectases, small pinkish spots, and atrophic white ones. There was a prominent wart the size of a pea at the outer canthus of the right eye, and in the centre of the left cheek there was a pea-sized scab, firmly seated, brown in colour, and surrounded by a raised edge, something like the rolled edge of epithelioma or rodent ulcer. The lips were dry and compressed. The conjunctivæ were slightly congested, the margin of the eyelids tending to ectropion. She was admitted into Ward 38 on 10th December. The wart on the left cheek had then fallen off, leaving a pink macular but superficial scar. Another had come in the centre of the right cheek, a hard warty growth with a narrow red areola. The urine was examined and found to deposit some urates, but contained neither sugar nor albumen. The treatment ordered was to have one quarter tabloid of thyroid extract daily, a small dose of cod-liver oil at night, and an ointment of boric acid in lanoline cold cream applied to the face. This seems to be but the second case reported in Scotland. Prof. McCall Anderson had one in a boy whose sister was also affected, though she died from an intercurrent complaint. One has also occurred in Ireland, figured and described by Dr Byrom Bramwell. There have been five in

England, described by Drs Radcliffe Crocker, Pringle, and Stephen Mackenzie. In all between sixty and seventy examples have been recorded from various parts since it was first brought under notice by Kaposi.

2. *Mr MacGillivray* showed—(a.) A case of EXCISION OF THE CONDYLES OF THE HUMERUS FOR DISLOCATION WITH FRACTURE. This was a case exactly similar to one he had shown to the Society a year ago, and was treated in the same way, with a similarly excellent result. The patient, a little girl aged 7, was sent in from the country, suffering from the result of a fall on the right elbow five weeks previously; ever since the joint had been fixed in a slightly flexed position. On examination, there was found to be a dislocation backwards and inwards of both bones of the forearm, with a fracture of the internal condyle, which was reunited to the lower end of the humerus, but displaced at right angles backwards. Reduction was impossible. He therefore, by means of a linear incision to the inner side of the olecranon, removed the condyles by the method recommended by Dr Heron Watson, by cutting off the internal condyle obliquely from above downwards by means of bone pliers, and removing it, separating the external condyle in a similar way from below upwards, turning out the end of the shaft and sawing it off, and then dissecting out the external condyle, the periosteum being left intact and the bones of the forearm untouched. No drainage tube was employed. Extension was applied for a week. Wound was then quite healed, and patient could flex the arm herself to her mouth; and now, three weeks after the operation, movement was almost perfect.

(b.) A case of a boy, aged 6, from whom he had removed EXTENSIVE PAPILLOMATA OF THE LARYNX BY THYROTOMY. This was the fourth occasion upon which he had operated for this condition, and so far as the operation was concerned it was perfectly easy and safe. He had formerly always done a provisional tracheotomy several days previously; but on this occasion the tracheotomy was done at the same time, a Hahn's tube being employed during the operation. The tracheotomy tube was removed the next day, and the whole wound healed at once. The trouble of the operation was the tendency to recurrence. There was no difficulty in thoroughly removing the warts from the vocal cords, but in every case he had operated on there were large bunches of warts growing from the ventricle of the larynx, and probably extending into the sacculus laryngis. These were extremely difficult to eradicate, and it was probably from these that recurrences took place. Although all difficulty in breathing was removed by the operation, he could not say that the improvement in voice was very great, the patient being only able to speak in a hoarse, toneless whisper.

3. *Mr Cotterill* showed—(a.) PANCREATIC CYST. Sarah B., aged 15

had suffered twelve weeks previous to her admission to Mr Cotterill's Wards from a sharp attack of gastric pain, accompanied by constipation and vomiting. A swelling began to show a fortnight after this, and at the date of her admission to the Wards there was a large tumour the size of a child's head in the left hypochondriac and umbilical regions, passing across the middle line to the right hypochondrium. This caused the patient a lot of dragging pain, and she was much emaciated. The exact nature of the tumour being doubtful, some of its contents were drawn off by the aspirator and examined, when the fluid was found to present several of the characters which were indicative of cyst of the pancreas. The chief interest in the case lay in the fact that the cyst was opened by a posterior incision, and not through the anterior abdominal wall, as is usually done. It was considered an advantage to attack the cyst from behind, as there it could be reached extra-peritoneally, it would drain better from a posterior opening, and there would be less risk of the subsequent formation of a ventral hernia. An incision was accordingly made along the outer border of the erector spinæ, just below the twelfth rib, and a tube was inserted into the cyst, the wall of which was found to be thick and nodulated on its external surface. The wound was kept open by drainage-tube for some four months, and latterly iodine was injected occasionally, to help to cause obliterative adhesions in the interior of the cyst. It was then allowed to heal, and has remained cured since that time. During her stay in the ward she was given liquor pancreaticus with her food, and this seems to have supplied a want, as she put on nearly 3 stones in weight in six months, and is now fat and well. The posterior incision was suggested to Mr Cotterill by a case of Mr Cathcart's, where he had found it necessary to make a counter-opening behind in a somewhat similar case, and had found that the wound drained well from the flank. The case shown is believed to be the first, in this school at any rate, which has been deliberately attacked from behind, and there seems little doubt but that it is the safer method.

(b.) LATERAL SPINA BIFIDA. Alexander A., aged 9, was sent in to Mr Cotterill's Wards on account of paralytic talipes equinus. On closer examination he was found to be suffering from a spina bifida, which protruded laterally through the sacrum through an opening opposite the right sacro-iliac synchondrosis. The tumour was about the size of an orange, and was covered by normal skin. It disappeared on pressure, and did not vary in size on coughing, but grew very tense when the patient stood upright. On passing the finger into the lower bowel it was found possible to invaginate the rectum through this bony aperture in the sacrum on to the posterior surface of that bone. Some fluid withdrawn from the tumour possessed the characteristics of cerebro-spinal fluid. The boy was found to be suffering from the following abnormalities in

conjunction with the spina bifida:—Imperfectly developed ears, especially the left, a very arched palate, ptosis of left eye, paralytic equino-varus of right side, congenital hydrocele, right inguinal hernia (congenital), an abnormally wide range of movement at the right shoulder joint, incontinence of fæces and urine, non-descent of the left testicle, and a somewhat backward mental development.

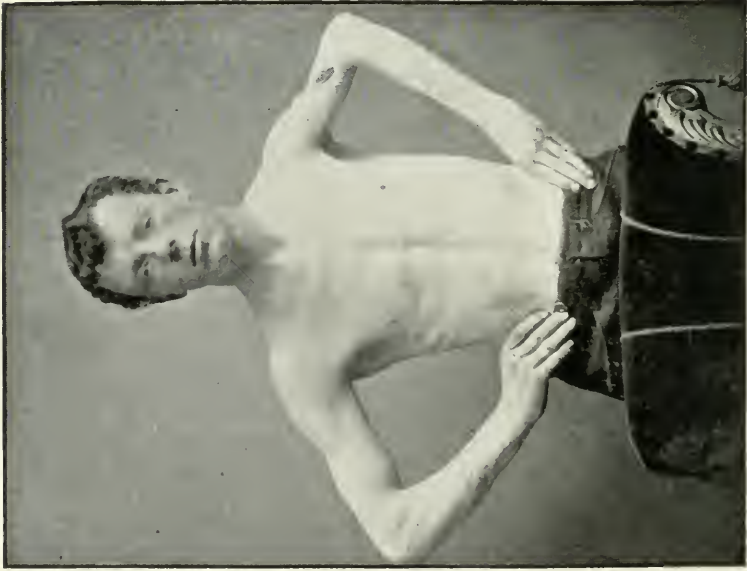
(c.) ACTINOMYCOSIS. Catherine F., aged 27, a domestic servant, was sent into Mr Cotterill's Wards suffering from a large swelling over the right zygoma and malar bone, of three weeks' standing. The symptoms had begun four weeks before admission by severe pain in the right ear, followed in a few days by swelling in front of the ear. There was no history of otorrhœa. Fomentations were applied for a few days, but as the swelling increased rapidly, an incision was made into it, and a few drops of pus evacuated, together with a considerable quantity of greyish pultaceous material, strongly suggestive of a rapidly growing sarcoma. It was discovered, however, to be a case of actinomycosis, the microscope detecting the ray fungus. The swelling was scraped with a Volkmann's spoon, and drained for two weeks, and iodide of potash was given in large doses. The patient is now convalescent (six weeks from the operation), the swelling having entirely disappeared, and the general health being greatly improved. Some decayed stumps were extracted, but no fungus was found about them.

In the above cases Dr Leith, pathologist to the Infirmary, kindly examined the specimens microscopically.

4. *Mr Caird* showed—(a.) A male patient, æt. 26, on whom he had performed a double (simultaneous) operation for PYLORIC STENOSIS AND DILATION OF THE STOMACH. The strictured pylorus was incised longitudinally and sutured transversely (method of Heineke and Mickulicz). The stomach was doubled upon itself, its greater curvature united to the lesser curvature, and the opposed serous surfaces to each other (method of Birchner). At the time of the operation, June 1895, he weighed 6 st. 12 lbs.; he now weighs 8 st. 7 lbs.

(b.) A lad, æt. 14, with a markedly tubercular history. He was admitted to the Royal Infirmary suffering from MIDDLE-EAR DISEASE, for which Prof. Chiene opened the left mastoid antrum. Three days thereafter he complained of severe headache, his temperature rose, and rapidly there came on incoherent speech, right side facial paralysis, twitching of right arm and leg, then twitching of all the limbs, succeeded by diminished motion, diminished resistance, and delayed sensation in the *right* limbs only. There was left optic neuritis. Temperature 104°. Under the belief that there was suppuration extending upwards from the Broca's convolution along the Rolandic areas, Mr Caird trephined. On opening the tense dura a quantity of serous fluid escaped; the brain bulged. There was no pus found on the surface of the brain, nor yet





TO ILLUSTRATE MR. CAIRD'S CASE OF WIRING THE HUMERUS FOR FALSE JOINT OF MANY YEARS DURATION.

within it on exploration. The pia was œdematous and showed a few milky patches along the vessels. On the following day the facial paralysis had passed off; the right limbs twitched more than the left; he was found to have right optic neuritis. Symptoms suggesting TUBERCULAR MENINGITIS now developed. He began to cry out, buried his head in the bed-clothes, his abdomen was markedly retracted, and *tâche cerebrale* was well marked. He became unconscious, and continued in an anxious condition for twelve days, when he began to improve. Optic neuritis attacked the right eye after the left had been affected, and the right was the last to resolve.

(c.) Patient, *æt.* 17, who, when 4 years of age, after an attack of measles, sustained an injury to the left humerus, which was followed by suppuration and exfoliation of the greater part of the diaphysis. A false joint ensued, and the stunted arm remained practically useless. After a couple of failures, the bones were successfully wired last year. Fragments of bone from the rawed extremities were left in the wound, and the process of consolidation was probably assisted by the employment of passive congestion a month after the operation. The arm is now strong and useful, but the whole of that extremity is much smaller than its well-developed neighbour.

5. *Dr John Thomson* showed, for *Dr Wm. Paterson*, a boy of 6½ years (M. H.), suffering from DEFORMITY OF THE HANDS AND FINGERS due to an attack of peripheral neuritis following measles. He had a severe attack of measles with pulmonary complications during the latter half of March. This pulled him down very much, but he seemed to have recovered pretty well. About the beginning of June he began to complain of tiredness and pains in his feet. Although the pains were never severe, his ankles gradually became extended so that he could not put his heels to the ground. His knee-jerks were gone. No rise of temperature. On 1st July, *Dr Paterson* found for the first time that there was distinct analgesia of the feet and legs. This passed off, and on the evening of the same day there was hyperæsthesia. There was also marked frequency of micturition. In the end of July, the feet were still so pointed that he could not stand, but there was no pain or tenderness. Early in September he was able to walk, and now his legs and feet seem quite right. On June 30th (a month after the feet) both his hands became sore in one day—with half an hour's interval. The pain was very severe, so that he screamed with it day and night for some days. It gradually subsided, but was still present in some degree four weeks later, and the hands remained very sensitive, apparently from hyperæsthesia of the skin as well as of the joint structures. The wrists became fixed in a flexed position; the fingers and thumb of the left hand were flexed towards the palm, the fingers of the right being extended at

the metacarpo-phalangeal joints and flexed at the others. (Photograph shown.) Attempts at extension were very painful. The position of the hands is slowly improving; they are very much less tender, and the range of movement of the joints is steadily increasing under regular passive exercises. The power of voluntary movement also is slowly returning to the fingers and hands. The prognosis seems quite good as to ultimate complete recovery.

6. *Dr John Thomson* showed a girl (*Jessie A.*), æt. 14, suffering from ELEPHANTIASIS AFFECTING THE NECK, SCALP, EAR, AND LOWER PART OF FACE. She was quite well until she was 3 years old, when her left cheek was noticed, during an attack of whooping-cough and inflammation of the lungs, to be swollen. No history of erysipelas can be obtained. The swelling has gradually increased in degree and extent. The left ear was not affected until some time after the neck. During the last three years the condition has spread considerably. (Photographs shown.) She is a tolerably well-grown girl, and presents no other abnormality. The lower part of the face—nose, lips, cheek—has its tissue distinctly thickened. The left auricle is much hypertrophied, and is fully  $\frac{2}{3}$  in. lower than the right one, owing to the swelling of the adjoining scalp and cheek. The left side of the neck is much swollen, the right side only slightly; on both there are a few hard, deeply-situated, enlarged glands. The mucous membranes of the mouth and throat are unaffected. Those of the nose and conjunctivæ are very prone to catarrh. There is a sore on the right cheek which shows little tendency to heal. The condition is precisely that which occurs sometimes after erysipelas. It is comparatively common in the extremities, but rare, I believe, in this situation.

7. *Mr David Wallace* brought before the Society a patient upon whom he had operated for CEREBRAL ABSCESS. *Jessie A.*, æt. 19 years, was admitted, on 15th July 1895, into the Royal Infirmiry, under the care of *Mr Caird*, in the absence of *Prof. Chiene*. For seven months she had suffered from middle-ear suppuration, but twelve days before admission the discharge from the left ear became more profuse, offensive, and of a green colour. On the evening of 14th July, when she returned from her work, she felt very tired, was sick, and could eat no supper. At times during the next day she was delirious, and her medical attendant advised that she should be sent to hospital. On admission she was restless and delirious; resisted examination of the ear, and did not recognise people. Pulse 56 to 64 per minute, weak and intermittent. *Mr Caird*, on examination, found a quantity of greenish putrid pus in the left ear—the membrana tympani was destroyed. There was fulness behind the ear, and pain on percussion over the mastoid. Temperature 101°.2. She complained of pain in the head, but did not localise it. *Mr Caird* opened into the mastoid antrum of the



left side, and from it a quantity of foul pus escaped. A free communication between the middle ear and antrum was formed. Next morning the temperature was 98°, but in the evening it rose to 100°·4; pulse 62, and respirations 28. After this the temperature gradually fell to below normal; the pulse became slower, falling on 19th July to 47; respirations, 28. In the evening the patient was observed to be drowsy, but answered questions; she was, however, more irritable than formerly. 20th July, 8 A.M.—Mr Wallace was asked to see patient, as Mr Caird was absent from town. She was found quite comatose; temperature, 97°; pulse, 44; respirations, 26. No symptoms had been observed which pointed to the cause of the condition, and in the first instance, therefore, Mr Wallace opened up the mastoid antrum more freely, but found no accumulation of pus. There was a sequestrum at the upper part, which, when removed, exposed the dura, which seemed healthy. It did not pulsate, and did not bulge into the opening. A large flap of the scalp was now turned back, and a circle of bone removed an inch above and an inch behind the auditory meatus. On exposure of the dura mater there was no pulsation, but after incising it the brain bulged into the opening. No pulsation was, however, present. An ordinary grooved director was now passed through the brain tissue in a downward and inward direction, when resistance was felt about  $\frac{3}{4}$  of an inch from the surface; with firmer pressure the cause of resistance gave way, and a quantity of extremely fetid sero-pus escaped. With sinus forceps the opening was widened, and thick green pus was evacuated, followed by sloughs of brain tissue—in all about 4 ozs. Mr Wallace introduced a finger into the cavity, and found that the greater part of the temporo-sphenoidal lobe was destroyed or pushed aside. A large tube was introduced, and the cavity gently syringed at low tension, after which the tube was fixed in position, and the opening in the mastoid packed with iodoform gauze. In the evening: temperature, 99°·2; pulse, 68; respirations, 28. 23rd July.—Tube removed; discharge very slight. Cavity practically *nil*. Piece of iodoform gauze introduced. 28th July.—Slight increase in discharge; a small tube introduced. 31st.—Tube removed and gauze inserted. During these ten days the patient gradually became sensible and able to answer and ask questions. Power in the left side was almost complete, but there was still marked loss of power in the right side. The eyes, examined ophthalmoscopically by Dr George Mackay, showed marked optic neuritis. 15th Aug.—Much better, but a little hysterical. 20th Aug.—Able to move about the ward. 24th.—Discharged, almost well. Still marked optic neuritis. There is slight amnesic aphasia, and slight want of power in the right leg. Mr Wallace said it was of great interest to note that in this case, *first*, marked optic neuritis was completely recovered from. Dr Mackay examined the eyes on 30th October, and found “the optic neuritis has passed off, leaving

but little trace of past existence." *Second*, That notwithstanding the large size of the abscess the patient had made an almost complete recovery. There was no paralysis and no headache. The only remaining symptom was a very slight degree of amnesic aphasia, which was gradually passing off. Indeed, now it was only when flurried that she experienced any difficulty in naming articles.

8. *Mr H. J. Stiles* showed—(a.) A child, aged 6 months, with an ARREST OF DEVELOPMENT OF BOTH EARS. The auricles, which were normal in position, consisted merely of a small unconvoluted reduplication of skin, apparently with no cartilage between. On the left side there were, in addition, two small pedunculated pear-shaped outgrowths, situated immediately in front of the rudimentary auricle. (In two instances within the last year or two *Mr Stiles* had removed a similar pedunculated excrescence from the same situation in front of a normal auricle.) There was no trace of an external auditory meatus on either side. The right half of the face was smaller than the left; the disproportion was greater as regards the lower jaw, the right half being much the smaller, so that the chin appeared twisted towards the right side. There was distinct paresis of the right frontalis muscle, and to a less extent of the right orbicularis palpebrarum. The right cornea had become opaque,—due, no doubt, to inability to completely close the eye. There was no paresis of the muscles of the lower half of the face. The mother had repeatedly observed that even very loud noises made no impression whatever upon the child. In other respects the child appeared to be in fairly good health, and evinced a fair amount of intelligence for his age. *Mr Stiles* referred to the futility of attempting anything in the way of operation, as it is well known that in such cases the deeper parts of the ear are also involved in the arrest of development. Through the kindness of *Sir William Turner*, he was able to show the Society an interesting specimen which had been presented to the University Anatomical Museum by the late *Prof. Allen Thomson*, and described by him in the *Monthly Journal of Medical Science*, vol. vii., 1846-47. In it the external auditory canal did not exist, in consequence of the tympanic plate being flattened up against the squamoso-zygomatic element of the bone. He also showed two specimens from a four-months' foetus which he himself had presented to the Anatomical Museum, illustrating the development of the malleus and of the lower jaw from *Meckel's cartilage*. Such specimens served to show how it was that the lower jaw was not infrequently involved when the ears are imperfectly developed. The paresis of the upper facial muscles was to be accounted for by an imperfect or compressed condition of the facial nerve which had to traverse the imperfectly developed temporal bone.

(b.) A female child, aged 18 months, with a WELL-MARKED

CERVICAL RIB on the left side. The mother did not discover the deformity until the child was some months old; on doing so she became alarmed, and took her to Dr James Murray, who kindly sent the case to the out-patient department of the Children's Hospital. The most striking feature on inspection is the high shoulder, with a corresponding shortening of the neck on the affected side, especially when viewed from behind. (It was from this aspect that the mother first noticed the deformity.) In front a prominence is seen above the middle of the clavicle. On palpation, a curved bony swelling is felt arching forwards above the clavicle as far as the outer border of the sterno-mastoid, where it appears to end in a free extremity. Posteriorly the bony swelling can be traced back to the spine. It has no connexion with the scapula. The pulsation of the subclavian artery cannot be felt; thus we may conclude that the vessel is lying anterior to the extremity of the cervical rib, and is not arching over it. Cervical ribs, although not infrequently met with in the dissecting-room, are seldom seen clinically, and it very rarely happens that the doctor is directly consulted on their account. He may, however, be consulted merely on account of the deformity, in which case the patient will most likely be an infant or young child. It is important, therefore, that the possibility of such a congenital abnormality be kept in mind, and the deformity to which it gives rise be recognised. In all, some eight or ten cases have been recorded (five during the present year—see paper by De Quervain, *Centralb. f. Chir.*, 1895, No. 47) in which the cervical rib has been excised in consequence of pressure upon the brachial plexus or the subclavian artery, or upon both. A case of cervical rib in an adult male patient was exhibited to this Society in December 1892 by Mr Wallace, who discovered the condition accidentally while examining the patient's chest at the Royal Public Dispensary.

(c.) A child, aged 5 years, with MULTIPLE AND SYMMETRICAL TUBERCULAR LESIONS, viz., in both elbows, in both supra-condyloid glands, and in the floor of both orbits, the latter situation being the seat of election for tubercle to attack the bones of the face. The early stage of abscess formation connected with tubercle of the floor of the orbit may readily be mistaken for a cyst. Its non-congenital origin and situation (over the outer part of the lower orbital margin) distinguish it, however, from an orbital dermoid cyst. The multiple and symmetrical character of the lesion in such cases is due, no doubt, to the entrance into the circulation of a quantity of tubercular débris, rich in bacilli, from some primary focus, the bacilli being suddenly showered down upon various parts, and taking root in those which are, or have been, rendered anatomically favourable to their further development. The same tendency to symmetrical location is not infrequently observed also in multiple acute abscesses.

9. *Mr Alexis Thomson* showed two patients upon whom he had operated for TRIGEMINAL NEURALGIA:—The first, a man of 74, had suffered for six years from attacks of epileptiform neuralgia, involving the left supra-orbital nerve in its terminal distribution on the forehead. The attacks were set up by opening the jaws. On the 30th April 1895, under cocain,  $3\frac{1}{2}$  cms. of the supra-orbital nerve were removed. The pain then shifted to a higher area on the forehead, in line with the point where the auriculo-temporal nerve crosses the zygoma. On the 12th May 1895, 2·5 cms. of the nerve were removed, also under cocain. Since then the pain has quite disappeared, and he cannot elicit it any more, however widely he opens the jaws. The scars are only recognised on close inspection. The second, a man of 55, had suffered for three years from very severe epileptiform neuralgia, involving the right half of the lower jaw and side of the tongue, and shooting upwards in front of the ear. Only temporary relief was obtained from large doses of morphine injected with the needle. It was decided to attack the main trunk of the inferior maxillary division of the fifth at the foramen ovale. This was carried out on the 10th July 1895 by the method associated with the names of Lücke and Sonnenburg, the zygoma being turned down with the masseter, the coronoid process reflected upwards with the temporal, the internal maxillary artery and its meningeal branch ligatured, and the external pterygoid muscle detached from the base of the skull until the trunk of the nerve was exposed at the foramen ovale. It was then divided, and its various branches followed for an inch or more and then divided one after another. The zygoma was replaced and sutured. Since then he has been absolutely free from pain. The scar on the face is barely visible. Although the lower jaw is stiff, he can chew meat unless it is tough. There is anæsthesia in the distribution of the nerves removed, and food may lodge between the cheek and the gum without his knowledge. He cannot shut the right eye by itself; otherwise there is no facial paralysis.

10. *Dr Norman Walker* showed—(a.) Four cases of LICHEN PLANUS. (b.) A case of LEUCODERMA. (c.) A case of ULCERATION OF THE TONGUE along with tubercular lesions on the skin.

## II. EXHIBITION OF SPECIMENS.

1. *Dr Burn Murdoch* showed—(a.) PATHOLOGICAL SPECIMEN from case of intestinal obstruction from impaction of a piece of raw rhubarb at a constriction in the small intestine. The constriction was caused by a puckering in of the gut, produced by a contraction of the mesentery, initiated by a few old-standing enlarged mesenteric glands. When first seen (twelve hours after the onset of the illness) the child was moribund. The symptoms were acute vomit-

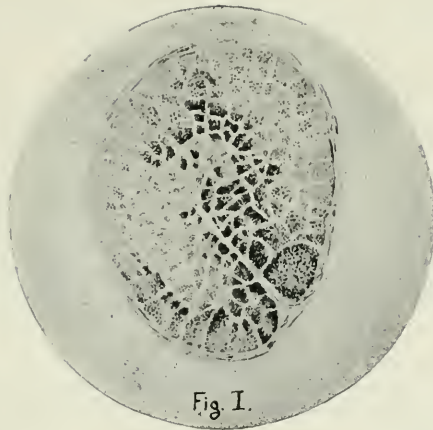


FIG. I.—Transverse section of optic nerve, stained with haematoxylin by Weigert-Pal method.

In the pale reniform area near the centre of the nerve, the nerve fibres were most degenerated. In the darker zone, which nearly surrounds this, there are fibres, some unaltered, but the majority showing a degeneration in an early stage, in which the altered myeline has not yet become removed from the nerve fibres, and therefore still retains the haematoxylin stain.

At the periphery of the nerve the stage of degeneration is intermediate between those described.



FIG. II.—Longitudinal section of posterior tibial nerve stained by Weigert-Pal method (oil immersion  $\frac{1}{2}$ ).

A, Axis-cylinder, varicose, denuded of myeline, sheath of Schwann partly stained.

B and G, Axis-cylinders with peculiar thickenings of myeline, perhaps in course of regeneration.

C, D, E, H, Axis-cylinders with much interrupted myeline sheath, often imbricated.

F, Commencing degeneration of the medullary sheath.

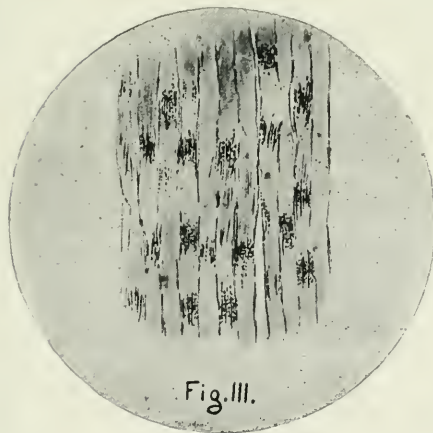


FIG. III.—Section of tibialis posticus muscle, Zeiss A, oc. 1, stained by Marchi's method.

This shows the peculiar disseminated areas of fat granules in the muscle fibres, with intervening fairly-healthy portions of fibre.

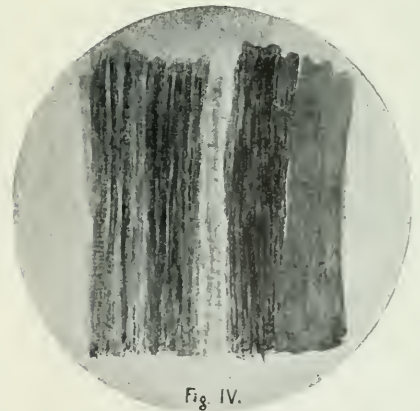


FIG. IV.—The same, under high power, Zeiss E, oc. 1.

The drawing shows the fine fatty granules between the fibrils, most of which have lost their transverse striation.

Note.—The figures have been reproduced from photographs of drawings by Miss J. M. MacGregor, M.B., C.M.



ing and purging. An interesting point in the history of the case was that the child, though otherwise quite healthy, had at times had attacks of colic, pointing to the passage of some article of food through the constriction.

(b.) PATHOLOGICAL SPECIMEN showing disease of the atlo-axoid articulation. The axis was eroded all round its pivot, and the atlas was diseased all over. At one side its substance was entirely gone, which accounted for the sudden death of the child, a dislocation having practically taken place, producing pressure on the spinal cord.

2. *Dr Allan Jamieson* showed the PARASITES OF RINGWORM, STAINED AND UNSTAINED:—(1.) Specimen of Sabouraud's trichophyton megalosporon as affecting the hair of a child's scalp, the spores within the root-sheath and inside the hair, the fibres of which were separated, and the texture broken up; unstained. (2.) The same form of fungus in a hair from tinea barbæ, the spores abundant in the root-sheath, the mycelium within the hair. (3.) The trichophyton megalosporon ectothrix from tinea barbæ, the mycelial threads ramifying round the outside of the hair; no visible spores, nor invasion of the hair. (4.) The same fungus stained according to the method described by Mr Malcolm Morris in the *Practitioner* of August 1895. (5.) Hair from ringworm of the head in a child affected with the trichophyton microsporon, the common form of ringworm fungus in this country and in France, but apparently not in Italy, where the megalosporon in one of its varieties seems more prevalent; unstained. (6.) Same fungus stained by Mr Morris's method, showing how the spores form an involueral sheath round the hair, while the mycelium which does not stain ramifies within the hair. (7.) Microsporon furfur; stained.

3. *Mr MacGillivray* showed—(a.) The CONDYLES OF THE HUMERUS which he had removed from the case he had previously shown, demonstrated the displaced inner condyle, and called attention to the large reproduction of new bone which had taken place through the operation having been performed subperiosteally.

(b.) The WARTS removed from the larynx by thyrotomy.

(c.) An OXALATE OF LIME CALCULUS he had removed from the ureter of a patient who had suffered for long from hæmaturia, pain in the back, and renal colic. The diagnosis had been made of probable calculus in the ureter from there having been an acutely painful pointed hard nodule to be felt from the front, a little to the right of the umbilicus. The incision was that for lumbar nephrotomy. The calculus was felt in the ureter, about 2 to 3 inches below the pelvis of the kidney; an incision was made into it, the stone removed, and three or four catgut sutures inserted. The urine was bloody for two days, and only then did

any urine escape from the posterior wound. The patient made an uninterrupted recovery.

4. *Dr Graham Brown* showed, for *Dr Mackenzie Johnston*, a SMALL METAL SHOE-BUTTON which had formed the nucleus of a rhinolith recently removed by him from a young gentleman 18 years of age. The rhinolith latterly caused bleeding and bad smell from the nose, and was about the size of a small grape. It seems probable that the button which caused this condition must have been about fifteen years in the nose.

5. *Mr David Wallace* showed—(a.) EPITHELIOMA OF LARYNX from case of laryngectomy. (b.) MALIGNANT DOUBLE STRICTURE OF OESOPHAGUS.

6. *Dr Leith* showed—(a.) SPECIMEN ILLUSTRATING COINCIDENT SIMPLE PERFORATING ULCER OF THE STOMACH AND PRIMARY COLLOID CANCER OF THE CÆCUM, WITH GREAT SECONDARY INFILTRATION OF THE DIAPHRAGM AND OMENTUM. Patient was a male, æt. 47, who first came under observation for a swelling of the abdomen. This was diagnosed to be malignant disease of the omentum, but the symptoms and signs were not definite enough to indicate whether any other organ was primarily affected. The post-mortem showed the cæcum to have been the primary seat of the disease. It was affected throughout its entire extent, its wall being much thickened and of a peculiar gelatinous or glue-like consistence and appearance, especially in its inner wall. No trace of the vermiform appendix could be obtained, the wall of the cæcum being considerably over 1 inch thick around its usual seat of origin. This may have been the point of origin of the disease. The lower lip of the ileo-cæcal valve was markedly affected, whilst the upper lip was free. The disease has mapped out the cæcum with great distinctness, as there is a sharp line of demarcation between it and the ascending colon. A perforation existed in the wall of the cæcum near the usual seat of the appendix, and had set up a septic peritonitis. There were over 2 pints of thick pus in the lower part of the abdomen and pelvis, roofed by the greatly thickened lower omental border and floored by the adherent pelvic structures. The peritoneal reflection of the ascending colon was greatly thickened, especially on the inner side, where it was over 1 inch thick, and extended upwards as far as the bend of the duodenum. The omentum formed a huge mass, greatly increased in thickness throughout its entire extent. It was about  $\frac{1}{2}$  an inch thick at its extreme right border, and over 3 inches thick at its left and at its attachment to the colon. It completely filled the abdominal cavity, except at its lower part, pressing the abdominal coils backwards. At the hepatic and splenic flexures it was continuous with the parietal peritoneum as it passed upwards beneath the diaphragm. This part of the parietal



peritoneum was over 1 to  $1\frac{1}{2}$  inch thick, and was adherent to the capsules of the liver and spleen. The other part of the parietal peritoneum was thickened. The peritoneum of the gastro-hepatic omentum was greatly affected, and formed a large mass, bigger than the closed fist. It had grown all round the lesser curvature and part of the anterior and posterior walls of the stomach, and at first gave the impression that it was really a tumour of the stomach. The mucous membrane was found, however, to be intact, and spread smoothly over the underlying tumour, except at one part, where there was a simple perforating ulcer upon the posterior wall about 4 inches above the pylorus. It was about  $1\frac{1}{2}$  inch long and  $\frac{3}{4}$  inch broad, and its floor was formed by the smooth capsular-like structure of the cancerous growth. It communicated with a suppurating sinus which passed for some distance into the cancerous omentum, and was also seemingly continuous with a small splenic abscess above the spleen. There was marked cancerous infiltration between the bladder and rectum. All the other organs were free from it. Microscope No. 1 shows the structure of the growth in the omentum. It is a typical colloid cancer of an advanced character.

(b.) TWO CASES OF MALIGNANT DISEASE OF THE TERMINATION OF THE COMMON BILE-DUCT. The first was in a male patient, aged 52, and the second also in a male some ten years older. In both there was persistent jaundice, but no tumour could be felt. In the second there was a definite history of gall-stones, but Mr Cotterill, after an exploratory incision, found nothing but a little thickening around or in the terminal part of the bile-duct. In both the lesion is closely similar, the wall of the duct in its last  $\frac{3}{4}$  of an inch being slightly thickened. In the first the lumen seemed smooth, but in the second it presented a distinctly granular appearance. There was more contraction in the first than in the second, and it gave the distinct impression that some cicatricial growth was present; but the appearances in the second did not lead me to suspect malignancy. The granular appearance on the surface I felt inclined to look upon as catarrhal, probably caused by a gall-stone. There was no trace of gall-stones in either case, but the ducts and gall-bladder were much dilated and the bile much inspissated. Microscope No. 2 shows that the first is a case of colloid cancer, and its remarkably small size and localization contrast strongly with the conditions seen in the last case. Microscopes Nos. 4 and 5 show that the second is a malignant adenoma in an early stage, the granular surface being due to the proliferation of the gland spaces. There is well-marked infiltration of the muscular coats.

The liver substance was markedly jaundiced in both cases, and there was a little biliary cirrhosis. Necrotic areas and abscesses were present in the first, shown in Microscope No. 3, and the liver cells were generally much destroyed in the second, shown in

Microscope No. 6, though no local necrotic areas or abscesses were present. Both cases illustrate well the destructive action which retained bile exerts upon the liver cells and the danger of a sepsis following thereupon, and are interesting also in the light of Stadelmann and Hunter's investigations into the irritating action of retained bile. It is possible that a poisonous or irritating substance is found even in such cases as the present. Both are powerful arguments in favour of a simple surgical operation being undertaken to draw off the bile, as thereby life may be considerably prolonged.

### III. EXHIBITION OF INSTRUMENTS.

1. *Mr Caird* showed a PORTABLE STERILISER FOR INSTRUMENTS.
2. *Mr Stiles* showed an improved TONSILOTOME (MATHIEUS).

### Meeting IV.—January 15, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. ELECTION OF MEMBERS.

THE following gentlemen were elected Ordinary Members of the Society:—William A. J. Alexander, M.A., M.B., 13 Gayfield Square, and William Tasker Lundie, M.D., M.A., B.Sc., 3 Glengyle Terrace.

#### II. EXHIBITION OF PATIENT.

*Mr Caird* showed a boy who had fallen from a height of one storey, landed athwart a barrel, and ruptured his urethra. All attempts to pass an instrument had failed. On the following morning his bladder was aspirated, and he was then sent from the extreme north of Scotland to the Infirmary, where he arrived late at night. The perineum was bruised, and distended with coagulated blood, but there was no evidence of extravasation of urine. Blood was passed from the urethra. *Mr Caird* thought there would be great difficulty in getting an instrument in. He cut down on a staff, but failed to find the urethra. Everything was such a mass of pulp that it seemed best to open the bladder above the symphysis. Having done so, and projected an instrument into the perineum, he then pulled through a red rubber instrument, stitched up the bladder, and then stitched up the external wound. For three days there was a slight urinary discharge in the dressing, but on the fourth day it had ceased, and they had union of bladder wound and of external wound by first intention.

## III. EXHIBITION OF SPECIMENS AND GELATINE CAST.

1. *Dr W. Elder* showed—(a.) The BRAIN from a case of old-standing aphasia. It was almost entirely a case of sensory aphasia. When the patient came under observation she was semi-comatose. The aphasia dated back to seventeen years ago, when she had a shock of paralysis about fourteen days after childbirth, in all probability of embolic or thrombotic origin. The paralysis involved the whole of the right side, leg, arm, and face, along with aphasia, but passed off very soon. In a few weeks it had almost disappeared, and in seven or eight months there was nothing left but aphasic symptoms. Nine months afterwards, however, mental symptoms showed themselves, and she was admitted to Morningside Asylum, where she was very closely observed, kept there for a fortnight or three weeks, and then sent to Larbert, where she recovered from her mental symptoms, but not from the aphasia. About five years ago she was again admitted to Morningside, having been found wandering about the street unable to give an account of herself, and was again carefully observed. Three years ago she had an epileptic fit, and three weeks before admission to Leith Hospital she had headaches and vomiting, and became comatose. She was admitted comatose, showing all the symptoms of a congestive attack so common in these brain cases. The symptoms shown in Morningside Asylum were limited almost entirely to the sensory side of the speech centres. She had paraphasia, *i.e.*, she could speak words, but could not converse intelligibly, used all sorts of words, but not the proper words. Her vocabulary as regards correct speech was limited to "yes" and "no" and a few simple expressions. She could not name her friends, husband, or the days of the week, but indicated them by signs. The hearing was perfect for sounds, but for words very imperfect indeed. She could understand the sentence "I am," but not "I am going out." She had also word blindness. She was previously a great reader, but since her attack could not read a book. She could recognise names of familiar friends in writing, but not such a word as "pain." She could check errors in butchers' and bakers' bills. A half loaf having been sent where a whole loaf was charged, she checked it at once. She could not sum up figures, although previously a good arithmetician. She was unable to write spontaneously, but could copy quite well. On her admission to the Asylum the facts stated on her certificate were that she was unable to give an account of herself, unable to give her name; had obstinate taciturnity; talked incoherently. The brain in this case was all normal except the first and second temporo-sphenoidal convolutions, which were almost entirely gone, completely atrophied, leaving only the lower temporo-sphenoidal. The marginal gyrus was slightly affected, thus explaining the

visual affection. She died from an attack of congestion, and the atrophied part was found full of clear fluid at the post-mortem.

(b.) A specimen of HYPERTROPHY OF THE BLADDER WITH DILATATION OF THE URETER, FROM STRICTURE OF THE URETHRA. It was very difficult to explain the dilatation of one ureter. It had possibly arisen from weakness in its wall or the lodging of a calculus in it, so starting the dilatation. Three small calculi were found in the bladder. The patient was admitted for uræmia, and died from it without giving any indication of this dilatation of ureter and pelvis. The ureter itself was very much dilated, but the pelvis did not appear to be dilated in proportion. It was rather difficult to make out where the pelvis ended and the ureter began. The kidneys showed consecutive Bright following a purulent condition.

2. *Dr Boyd* showed a specimen of ANEURISM OF THE AORTA WITH RUPTURE INTO THE LUNG. The patient got up one Sunday morning apparently in perfect health, but was afterwards found half out of the bath and bleeding from the mouth. When got back to bed he was dead. He (*Dr Boyd*) made a post-mortem, and on opening the chest found the whole of the left pleura adherent. There was an enormous aneurismal dilatation of the aorta. At the descending part of the arch the aneurism had perforated through the two layers of the pleura into the lung, and there was a small hole leading into a bronchus. There was very marked atheroma of the whole of the aorta. The peculiar points were, first, that the patient had absolutely no symptoms, was apparently in perfect health up to the time of death,—he had not been seen by a medical man for fifteen years; secondly, the small amount of hypertrophy of left ventricle in spite of a certain amount of aortic incompetence; and, lastly, the adhesion of the whole of the pleura without any symptoms, and the aneurism passing through the two layers into the lungs without any sign. There was no history of syphilis.

3. *Mr Greig* showed a GELATINE CAST OF A CASE OF LUPUS VERRUCOSUS. The cast had not been taken on account of any inherent peculiarity in the disease itself, but simply because he thought it interesting to try whether it was possible to bring out the absolute detail, as *Mr Cathcart* claimed. The case presented many points of interest. It was an affection of the forearm and hand of a female, aged 11, whose family history was good. She stated, and her mother corroborated the fact, that the disease had lasted for about five years, and she thought it had commenced on the back of one of her hands. It was worse in winter, better in summer. She had been in Dundee Royal Infirmary twice; and to *Dr Stalker* he was indebted for permission to take the mould. The rash was most marked on the dorsal aspect of hands, wrists, and forearms. There was a patch of lupus verrucosus on either

cheek, also on dorsum of both great toes, inner borders of both feet, inner aspect of heels at insertion of tendo Achillis, and on an area over the patellæ, on inner aspect of each knee, and above and below the gluteal folds,—on the latter situation quite small in amount. The cast showed other details of the case worth noticing. First the skin itself presented a dull opaque condition with a considerable amount of scaly epithelium, more like a branny desquamation. All over the affected limbs there was an eruption of small papules very well shown on the cast. These were pinkish, and many of them had their tips rubbed off or broken down, probably not due to scratching, but simply to friction against clothes. Around the verrucose areas the skin was of unnatural pinkness, shown very distinctly, but not exaggerated, on the cast. Certain parts on the dorsum of the hand were raised, and formed the verrucose patches which gave the disease its name. On other parts of the limbs the verrucæ had a peculiar flattened form, or formed a desquamating, slightly raised areola round about a more or less cicatricial area. These were the points worthy of notice which he thought he might claim that this "Cathcartine" cast showed very distinctly.

#### IV. ORIGINAL COMMUNICATIONS.

1. *Dr Noël Paton* read his paper on THE ACTION OF THE LIVER ON FATS, of which the following is an abstract:—

Dr Paton pointed out that the old idea of the liver being simply a digestive gland had been exploded by Bernard, and that all subsequent work had confirmed his conclusion as to the relationship of the liver with the metabolism of the carbohydrates. Its connexion with the metabolism of proteids had also been demonstrated. But while the liver is in the channel of absorption of these substances, it is not in the channel of absorption of fat. Has it, then, any special connexion with the metabolism of fat?

At present evidence on this point is wanting, and the object of these observations is to elucidate the subject. The want of information is probably due to the tedious character of the experiments required and to the difficult and lengthy analyses involved.

The method of estimating the liver fats was first considered, and it was pointed out that the ether extract contains only about 57 per cent. of fatty acids,—the essential constituent of the fats,—instead of the 90 or 92 per cent. found in the ether extract of adipose tissue. Hence all conclusions based upon the mere estimation of the ether extract are valueless. The fatty acids must be determined.

Various questions of purely physiological interest were passed over, and attention was next directed to the chief combination of the fatty acids in the liver—Lecithin. Its chemical nature was considered, and its relationship to phosphorus pointed out. It

constitutes on an average 2.35 per cent. of the liver and about 10 per cent. of the solids. About half of the fatty acids are normally combined in this substance. The significance of its occurrence in such large amounts was considered. The part played by lecithin as the source of phosphorus for the bone and nucleins of the growing embryo was pointed out, and it was submitted that the lecithin of the liver performed a similar function, combining with phosphorus as a first step to the formation of nucleic compounds.

The influence of different conditions on the fatty acids of the liver was next dealt with, and it was shown by experiments on kittens and pigeons that the normal average amount of fats was not diminished even after four days' starvation. The question of why the store of fat should be maintained in the liver was considered, and it was suggested that its purpose is to combine with the phosphorus produced by the decomposition of nucleins, and to prepare it for use again in the protoplasm.

It was shown by experiments of feeding kittens upon cream that the fats are stored in the liver, and that the excess of fats is only got rid of after about sixty hours.

Experiments were also described which indicated that carbohydrate food increases the fats of the liver. The possible formation of these fats from glycogen was considered, and it was shown that as the glycogen disappears there is an *actual* increase in the amount of fats. Since this is an increase of the characteristic fats of the liver, and since the blood serum manifests no milkiness, the fats would appear to be actually formed by the liver protoplasm from glycogen. This does not exclude the possibility of the liver also forming sugar from the same source.

The question of fat formation from proteids was discussed, and it was shown that proteid food did not lead to an accumulation of fats in the liver.

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*The President* said he would be glad to hear members speak on this communication, which dealt with a subject on which great diversity of opinion existed, and the nature of which remained, he might be allowed to say, a little obscure. He would be glad to hear those who could speak with authority on these points.

*Dr Gibson* said that he made no professions at all in the matter of physiological chemistry, but thought there were a great many points in this paper of interest to the practical physician. He asked *Dr Paton* how, if lecithin, built up as it was, were stored in the liver, there should be such a tremendous disappearance of the tissues of the liver in cases of phosphorus poisoning. No doubt there was storage for the time being of fat in the liver, but only as a half-way house. With regard to the storage of fat in the liver, *Dr Paton's* observations ran counter to some of the teaching which had almost become

current coin, *i.e.*,—the teaching of Oertel and others, who made it their duty to restore a graceful form to those unfortunate members of society affected with a tendency to obesity. He almost feared, in the present state of matters, to say anything at all about the relation between the carbohydrates and the fats of the liver, because, if Dr Paton would allow him to say so, owing to the strained relations between London and Edinburgh on such subjects, it was rather a dangerous subject to take up at all. He had always believed that from the carbohydrates which were absorbed fats were formed and deposited, and Dr Paton had brought before them facts that certainly proved that point; but in regard to the relationship between the storage and absorption of glycogen and the transformation of carbohydrates into fat going on in the liver, he thought perhaps the practical physician might be a little aided if Dr Paton would in his reply give them one or two more practical deductions. The tables shown by him had passed too rapidly for them to catch all the facts. The only other point he might refer to was that his observations with regard to the relation between proteids and the storage of fats in the liver amply confirmed what in practical life they had been long accustomed to, —*viz.*, that out of proteids there was but little fat absorbed and deposited. Such were the points that from the point of view of the practical physician emerged from this paper, and he would only like to express the extreme gratification and pleasure with which he had listened to it.

*Dr J. C. Dunlop* said they had listened to a vast amount of new material in contradiction to the text-books. What struck one most was that the amount of lecithin was vastly greater than all previous statements, and also the effect of hunger. He (*Dr Dunlop*) had made observations on the quality of fat to be met with in degeneration and infiltration, but could not find any essential difference between the two. He found in three fatty infiltrated livers that the melting-points of the fatty acids were 8, 27, and 34; in two degenerated livers, 16 and 27. It seemed there was no chemical difference between the fats of the two diseased livers.

*Dr Lovell Gulland* said there was only one point in *Dr Paton's* paper that he wanted to draw attention to, because most of it was really far beyond any one who was not a physiological chemist. It seemed to him that the interesting point, so far as he made it out, in *Dr Paton's* paper was the rôle of the liver in keeping a balance of phosphorus for the nuclear compounds of albumen. It was a suggestion which was new to him (*Dr Gulland*), and, he thought, was new altogether. One would not have supposed that the liver, whose energies, one would have thought, were fully taken up in other directions, should have laid itself out to store phosphorus for the nuclei and cells of the body, and it seemed to him that, if *Dr Paton's* suggestion would hold, it would be a most fruitful field for

further investigation, and he foresaw that there might possibly even be histological developments, which would be of exceedingly great interest. He hoped Dr Paton in future researches would leave the beaten track of carbohydrates and achieve something in this direction.

*Dr Gillespie* said that Dr Paton had mentioned that in starvation the phosphorus in the liver was not diminished. He (Dr Gillespie) wondered if he had investigated the amount of phosphorus passed in the urine at the same time. It appeared to him that if the amount of phosphorus remained constant in the liver and there was some loss in the urine all the time, the bones and other parts of the body containing phosphorus must be losing phosphorus, which was kept up to the normal in the liver. He thought it was Dr Pavy, Dr Paton's friend in London, who stated that carbohydrates might be turned into fats in the cells in the walls of the intestinal villi. He would just like to ask Dr Paton—who he knew denied it—assuming it to be a fact that fat was formed in these villi, what effect it would have on his researches on the fats of the liver?

*Dr Stockman* said that with regard to the fattening experiments, Dr Paton had brought before them a number of tables showing that the melting-point diminished when they gave such things as cream. He (Dr Stockman) wanted to know if he thought cream was absorbed as such, and was not much changed in the body; because, if so, when one started fattening a person on cream one would get a white, creamy fat. If, on the other hand, cod-liver oil were given, there ought to be a yellow fat. It seemed to him (Dr Stockman) that the fat must undergo some change, and not be simply an infiltration, and that it could not affect the melting-point so directly as Dr Paton said. One other point he would like to ask him about. He understood him to say that the glycogen formed fat, but he understood that the point at issue between him and Dr Pavy was that it formed glucose entirely, and not fat. He would like to know also if the fat cells did not play an important part, or were entirely passive. (Dr Paton: "You mean adipose tissue. Undoubtedly it does. I pointed that out.")

*Dr Paton*, in replying, said he would be as brief as possible. Phosphorus poisoning he declined to enter upon. It raised so many vexed questions that he did not discuss it. Dr Abernethy was at present working at it. It actually diminished the lecithin. As regards the giving of fatty food for obesity, the results might be obtained by inducing dyspepsia and diminished absorption. Dr Gibson had asked if he could draw any practical deductions. He thought common experience had drawn these practical deductions before physiologists investigated the question. Dr Dunlop's remarks were of great interest. He (Dr Paton) expected different results. They did not know the diet of the patients from whom the liver had been taken. One might have been taking cod-liver



oil, another beef-fats. As to Dr Gillespie's remarks, in starvation phosphorus fell rather less in proportion than urea. As starvation was going on there must be a certain loss. All he maintained was that the liver interfered with the loss. As to the fat in the cells of the intestine, Dr Gulland and he had investigated this thesis of Dr Pavy's, and he thought they managed conclusively to prove that the carbohydrates did not form fats in the cells of the intestine. What Dr Pavy found was the fat in the oats on which he had fed his rabbits. Dr Stockman raised the question of the changes of fats in absorption. It had been shown that fats of lower melting-point were absorbed more rapidly than those of higher melting-point. There was no doubt, from recent researches, that after the administration of rape oil fats there was a large amount in the tissues, and undoubtedly in patients who had been taking cod-liver oil there was an accumulation of fat, with a large amount of oleic acid. While he thought there was evidence that glycogen could be converted into fats by the liver cells, that by no means disproved that the liver cells could also form sugar from glycogen, that fat and sugar were products of liver cells, which could form them out of anything it could get hold of for the purpose.

## 2. AN INQUIRY INTO THE PHYSIOLOGY OF THE ACTION OF THERMAL SALINE BATHS AND RESISTANCE EXERCISES IN THE TREATMENT OF CHRONIC HEART DISEASE (THE NAUHEIM AND SCHOTT SYSTEM).

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THIS method of treating chronic heart disease, commonly called in this country "the Schott System," in recognition of its practical discoverers and exponents, was lately brought into prominence in England by Dr Bezly Thorne, and is now attracting much attention. It is claimed for it that it is applicable to an immense number of cases which hardly improve, if at all, under rest and ordinary cardiac tonics, and yet are not strong enough to be able to adopt the more vigorous measures of the Oertel System; that, in fact, it has discovered a path of safety intermediate between the Scylla of over-inaction and the Charybdis of over-exertion, which leads gradually and surely to the re-establishment of sound cardiac and bodily health; that its mildness renders it almost universally suitable; and that its beneficence and power, when employed by skilful hands, are capable of accomplishing results alike comforting, reassuring, and delightful to the patient and gratifying to the physician. If such claims can be substantiated, if they are found

to rest upon a sure foundation, and if they are supported by sound physiological and clinical proof, they will most assuredly be welcomed by us with gladness. We have been taught by experience, however, to walk warily. We recall to mind that such sanguine views, amounting in many cases to exaggeration, are common to every new health-resort,—whether it be a spa more remarkable than all others for the unpleasantness of its waters; or an air cure, in which the credulous go barefooted upon the dewy grass or snowy pavements; or the whey cure, where they swill fermented milk to a perilous stage of repletion; or the grape cure, where they eat of the juicy fruit to a degree far beyond the dictates of common-sense or the laws of digestion; and all this too, be it remembered, under the specialist's direction. Health-resorts are as much subject to fashion as dress or music, or bicycling or serving a dinner, and the medical specialist is too apt to follow the prevailing craze of the time, and recommend the latest favourite spa. The fascination of trying a new remedy and the certainty of pleasing his patient tempt him, and he saves argument and his time, already perhaps overtaxed, by adopting what is often the obvious wish of his patient. A particular "Kurort" may thus gain a reputation far above its merits. There is a perfect library of literature on spas and other places, that possess in baths, waters, or climate those sovereign cures of which sooner or later the majority of mankind are in search. There is scarcely a disease from ennui to gout which has not its appropriate waters, saline and sulphurous, earthy and ferruginous, alkaline and acidulated, indifferent and uncharacterised; and the cautious and critical mind lends a very guarded ear to claims coming from sources so liable to exaggeration and over-pretension. Bad Nauheim claims to possess in its warm saline carbonated waters a sovereign remedy for chronic heart disease, and its praises are voiced by thousands of the laity and its claims supported by medical men of more or less eminence in nearly every country in Europe. There are the many writings of the two brothers Schott and of Bezly Thorne, and the papers of the two Broadbents, of Babcock, of Israel, of Moellier, of Groedel, of Saundby, and others, all testifying more or less strongly in their favour. They have, however, mainly devoted themselves to an account of the waters and the resistance exercises, their methods of use, their effects, their artificial reproduction at home, and the rationale of their action, on the assumption or conviction that the claims advanced are well founded. This seems to me to be neither satisfactory nor scientific; it is surely better to subject these claims themselves to criticism, and to test them in every way possible. Balneological methods in the past have largely erred in a want of exactness of technique and physiological grounds for their action. The first objection can hardly be urged against the Schott System, as the composition of the baths, their method of administration, the conditions under

which they are to be taken, and the precautions to be adopted, have all been given with much care and precision by Dr Aug. Schott, and subsequent writers on the Continent and in this country have all adhered more or less closely to his views. The same may be said of the exercises. When we come, however, to study the physiological explanations of their action, we have hardly the same cause for satisfaction. The results of their action are grouped as follows:—

I. *Subjective Phenomena.*—1. A sensation of cold followed by warmth. The patient lies in the bath without movement, and the sensation of slight cold experienced on entering is soon replaced by one of gentle warmth, which lasts during the remainder of the bath. The same experience occurs, but to a slighter degree, with each succeeding bath, so that the patient is able comfortably to bear a gradually diminishing temperature, until he reaches the coldest baths usually administered, viz., the carbonated natural springs, about 86° F. In these the gentle tingling, warm sensation all over the skin is particularly marked.

2. A sense of oppression in the chest. This gradually becomes less marked after the patient has been some little time in the bath, and usually diminishes in subsequent baths until it may become hardly noticeable.

3. An increased power of sleep. It may become more regular, constant, and refreshing, and troublesome insomnia may even disappear.

4. Disappearance of unpleasant sensations. If the patient has formerly suffered from palpitation, cardiac pains, breathlessness, or headache, he frequently finds that they disappear. He is, further, conscious of an increased sense of wellbeing. He is lighter in spirits and fitter for work, and generally expresses himself as feeling much better.

II. *The Objective Phenomena.*—1. An increase in strength and diminution in rate of the pulse. The extent of the change varies in different cases. A diminution of six to ten beats in a minute is a fairly common average, and while the increase of strength is never great, it is easily appreciated by the fingers. Other changes are also occasionally observed,—*e.g.*, irregularities in strength and rhythm may be modified, and may even disappear.

2. Slight slowing of respiration. This usually occurs to the extent of two to three respirations per minute during the action of the remedial agent.

3. A diminution in size of the heart. This is the main apparent result, though inspection, palpation, and auscultation may also give evidence of an improved heart tone. Thus a systolic mitral or tricuspid murmur may disappear and a presystolic mitral reappear, or the cardiac sounds may improve and the apex-beat become more forcible; but all these changes occur to a slight degree only, and sink into comparative insignificance beside the great diminution

which takes place in the area of deep percussion dulness in the cardiac region. The extent of this diminution varies in different cases, but  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch all round is quite common, and in many cases it even amounts to 1 inch or more. It is held that this diminution of the area of cardiac dulness indicates a corresponding decrease in the size of the heart.

I have now made many observations upon the working of The System, having studied it carefully at Nauheim last summer along with my friend Dr Frederic Thorne of Leamington, and afterwards by means of the exercises and artificial baths at home. The number of cases already reported on by different writers as having benefited by the treatment is already very great: thus Beneke in 1872 gives 56; Aug. Schott, writing in 1885, gives over 300; and his brother, Theodore Schott, in 1894 gives about 4700; while Bezly Thorne and others have also added considerably to the number. I cannot claim to possess so lengthened a period of experience of the System as some of these writers, nor do I think it is necessary to enable the inquirer to test it thoroughly, or to judge of its merits or demerits with impartiality. My experience has enabled me to study several examples of different classes of cases, which may be conveniently grouped as follows:—

I. *Cases of Functional Disease.*—1. Post-influenzal.—The symptoms due to the legacy of influenza varied considerably: there was bodily and mental weakness, pain or discomfort, referable to the cardiac region or its neighbourhood, giddiness, headache, palpitation, or gastro-intestinal derangement. The pulse was generally fast and soft, and sometimes irregular. The area of percussion dulness in the cardiac region was apparently mostly increased in varying degree, being in a few cases considerably so. In none was a murmur present. 2. Overstrain.—They were apparently referable to overwork or mental worry. In some it appeared to be an acutely produced condition, as the result of too vigorous bodily exercise after a prolonged period of rest. In others it seemed to be of more gradual production. 3. Neurotic.—This is a variable group. In some the disturbance was probably of primary central origin, as in people of high-strung, sensitive natures, sometimes with an undue perception of their own heart's action, while in others it was apparently referable to an abuse of tobacco, and in others to a peripheral stimulation of the vagus from a severe gastric or intestinal catarrh, which was the beginning of the illness. 4. Anæmia, etc.—They showed general deterioration of the system, anæmia, loss of energy so characteristic of prolonged town life, etc.

The symptoms and signs in these three latter groups were somewhat similar to those of the first,—breathlessness, even amounting to dyspnoea, or a sense of suffocation on exertion, being more frequently complained of; tenderness in the region of the liver, inability to lie on the right side, disturbed sleep, fulness of the veins of the root of the neck, pulsation in the second left inter-

space just outside the pulmonary area, or in the third, fourth, or fifth intercostal spaces immediately to the left of the sternum, or in the epigastrium, and systolic murmurs, were occasionally observed.

The general effect of the therapeutic agents, baths and exercises, upon the majority of these cases was much the same. Their general condition was improved, many of the troublesome symptoms disappearing altogether. After each administration the average diminution in the pulse was 6 to 7 beats, the maximum being 15, and the minimum 0. The average reduction in the area of cardiac dulness in the fourth interspace was apparently  $\frac{5}{8}$  to  $\frac{3}{4}$  inch, the maximum being  $1\frac{1}{4}$  inch, and the minimum practically *nil*. The transverse diameter of this area in these cases varied between 4 and 7 inches. The reduction was generally greatest on the right side; thus in two cases of 7 inches there was an apparent diminution of nearly 1 inch on the right side (from 3 to 2), and of about  $\frac{1}{4}$  inch on the left side.

II. *Cases of Organic Disease.*—1. *Valvular Lesions.*—Some were of rheumatic, and others of apparently spontaneous origin. In one case the disease had been in existence for over ten years, and in another for over twenty; and some of them were fairly good illustrations of the evils of venous congestion, œdema, etc., which follow upon loss of compensation.

The average diminution in the pulse was 8 beats, and reduction in the area of cardiac dulness  $\frac{1}{2}$  to  $\frac{5}{8}$  inch, measured in the fourth interspace. The primary extent of the dulness in this interspace varied between  $7\frac{1}{2}$  and 5 inches, and the left side was, if anything, more diminished than the right. They were generally improved in health. One, a doctor, with mitral disease, wrote me about five weeks after the end of his course, that he was better than he had been for years; and again, about a month ago, to the effect that the improvement had partly disappeared, but that he was still better than before his course of treatment.

2. *Congenital Heart Disease.*—I have seen patients who have greatly improved under treatment, but never anything to indicate a disappearance of the primary lesion.

III. *Cases of Simple Angina Pectoris and Graves' Disease.*—I have hardly seen a sufficient number of these cases to enable me to give definite data, but, generally speaking, I am able to corroborate the experience of others, that improvement does occur in some of these cases.

My experience of the System has undoubtedly very favourably impressed me. It has enabled me to corroborate its claims to be regarded as a therapeutical agent of considerable value; but while doing so in general, I am unable to do so in all its details, or to support some of the most striking claims it has put forward.

Before proceeding to discuss the pulse and heart, I may glance for a moment at the subjective phenomena which are pointed to—

and rightly, I think—as excellent evidence in its support. The patient's own notions regarding his progress generally receive our due attention. If he believes he is recovering, he is not so very often wrong. I was much struck by the remark made by a scientific friend, a learned and deservedly eminent professor, who was himself undergoing the treatment, in the course of a conversation upon the method, to the effect that the doctors might not like or approve of it until they understood it, but that at present the unfortunate patients themselves did like it, as they found themselves benefited by it. They have good reason for this if they can point, as they often can, to the disappearance of symptoms which may have formerly made their lives miserable.

We accept these statements and value them, but it is a different thing when we come to base physiological arguments upon them. Thus it may be true—nay, it is true, as I have myself verified in my own person—that the first sensation of cold on entering the bath is quickly replaced by one of warmth, especially in the carbonated waters, but it does not necessarily follow, as so many suppose, that this is due to a definite nervous stimulation. Indeed, the primary difficulty of relying upon sensations experienced under water at once occurs to us. The personal factor, which depends largely upon the state of the circulation, the activity of the sweat secretion, the excitability of the nervous system, and the habits and customs of the patient, has to be carefully taken into account, and it is next to impossible to properly appreciate any factor so variable. What is cold to one man is warm to another. I may mention the remark I heard made by a distinguished colleague in Edinburgh, in the course of a conversation upon the value of a cold bath in the morning, as a somewhat striking illustration. In referring to his own habit of taking a cold bath all the year round, he said he always filled his bath three-quarters full from the cold tap, and then lay down and remained in it until he got warm. It would hardly be wise for us to advise all our patients to follow this Spartan example.

I shall defer for a little the further consideration of the influence of the temperature, pressure, and  $\text{CO}_2$  of the water upon the vascular and nervous mechanisms of the body, and pass on at once to the objective phenomena.

In referring to the objective phenomena I have already pointed out the important part played by percussion in estimating the effects of the treatment. Indeed it is customary to refer to the diminution observed in the area of cardiac dulness after each application as an indication of the progress made by the patient, and charts have been freely published under the conviction that they truly represent the area occupied by the heart before and after treatment. I have never been able to convince myself that this is really the case. There are too many difficulties in the way. In the first place, I do not believe that percussion of the

heart can be made with anything like sufficient accuracy to meet the requirements of this assertion. I was much impressed with the difficulties I have frequently met with in outlining the cardiac area. In some cases it seemed, in part at any rate, easy enough; in others it was impossible to come nearer than an approximate guess. This is especially the case on the right side, and in the upper part of the sternum. I am certain that the experience of others will bear me out in this. Indeed, when working, as I was many times privileged to do, along with other and skilled clinicians, I have observed the same uncertainty in them, and it was the rule and not the exception to find considerable variations of opinion as to the exact cardiac boundaries in any individual case. I had often previously experienced this difficulty in ordinary clinical work, but it had not impressed me so strongly as it now did, when percussion had to be made so frequently, and played such an important part in the diagnosis, prognosis, and progress of the cases. I was led to pay special attention to it. I outlined the heart by percussion, in a great many cases, on the cadaver, and thereupon compared this area with that actually occupied by the heart. The cases selected were representative of the different ages, different thicknesses of thoracic wall, and depth of cavity. I found the percussion outlines to be almost invariably nearly accurate, the different characters of chest, etc., presenting no real difficulty. Extensive emphysema of the lungs, or cases of considerably dilated right heart, where it was covered deeply by lung, did, however, present some difficulty, especially on the right side, the percussion line erring sometimes as much as 1 inch to 2 inches.

Ordinary percussion difficulties must, therefore, be chiefly due to conditions prevailing during life. So far as we can see, these ought to be one or more of the following factors: the movements of the heart itself, of the lungs, of the stomach and liver, the vibrations of the sternum, and the elasticity of the other tissues or those characters peculiar in some way to their vitality, and so far unknown to us. The ordinary systole and diastole of the heart are so rapid and short-lived that they cannot really affect percussion. By taking proper precautions the influence of the stomach and liver can be readily eliminated. This influences the vibrations peculiar to the sternum; and those, if any, attributable to the other tissues, while they materially alter the percussion note and make its correct appreciation more difficult, can hardly be held to change much during any moderate interval of time at any rate. There remains, then, apparently only the movements of the lungs. Any one can readily satisfy himself that they may materially alter the area of cardiac dulness. I have made several observations on this point in healthy adults, and have found wide variations. I have found the area to be reduced by nearly one-fourth in its transverse diameter in the fourth interspace, by directing the individual to breathe more

deeply than usual for five minutes continuously. There are considerable individual variations in this respect, depending upon the configuration of the chest, the respiratory habit of the patient, and perhaps the size of the lungs themselves. In some, even a few deep inspirations, or a single one, materially diminish the area. I am of course speaking of the deep cardiac dulness, and yet no one would for a moment contend that these differences represented actual diminutions in the size of the heart. If these observations be correct, it follows that percussion cardiac areas lose their reliability as indices of the size of the organ, unless always obtained under precisely similar conditions of respiration and lung expansion. Trivial actions, *e.g.*, a few minutes' gentle exercise, smoking a cigarette, even change of posture, seemed to have an appreciable influence. I need not, therefore, point out how difficult it is to secure such conditions; indeed, I found very considerable variations in percussion of the same healthy individuals on each successive day, always under what I judged to be similar circumstances. Some apparently showed little variation, others a good deal. I had the same experience when I tested them at different times upon the same day. Lastly, I compared the results obtained in the same individuals (four healthy males) several times within fifteen minutes, they sitting the while on an easy chair, and found that two of them varied decidedly nearly every time, and the other two slightly. I carefully marked the position of the apex-beat each time in all the cases, and found that in most it remained fairly stationary, although the area of cardiac dulness sometimes showed a good deal of change, while in others there was a slight degree of displacement, amounting even to half an inch. I was considerably puzzled by these results, as they seemed to indicate changes other than those attributable to the lungs, and if so, they could only be referred to the heart itself. It looks as if the heart were capable of considerable movements other than its ordinary rhythmic contractions, which affect the apex less than any other part of it. When discussing this question with Dr George Balfour, he mentioned a paper by Heitler of Vienna as likely to interest me, on "The Percussion Conditions in the Normal Heart." He afterwards kindly sent it to me. It contains the results of much careful observation. He dwells upon the very varying boundaries given by writers for the right, left, and upper boundaries of the healthy heart, obtained by percussion, and points out that the wide differences of opinion probably arose from the difficulties in the way of obtaining accurate percussion areas. He gives the results of his own observations upon healthy adults. He holds that the area of cardiac dulness is constantly varying; that as the observer percusses over the anatomical cardiac area from without inwards, or in the opposite direction, or vertically, he finds the dulness disappearing under his finger, to reappear again immediately; that any spot in this



area is at one time dull and the next clear; that, in short, the whole cardiac dulness disappears and reappears with peculiar regularity. It appears mostly four times, less seldom three to five times, in a minute, and fades away gradually. Its duration is from two to five or six seconds. He attributes these disappearances to actual changes of volume in the heart, which do not affect the pulse, and therefore holds that, to be of any value, percussion of the heart must be always made during the phases of full expansion. These observations of Heitler explain the variations met with by me in normal individuals, and I am inclined to place considerable value upon them. Since reading his paper I have made a number of further observations upon the percussion areas of the healthy heart, and while I cannot say that I have been able to fully verify his statements, I have obtained results that I am really unable to explain except upon some such hypothesis as he has put forward. The difficulties experienced in percussion of the normal heart become greater, and not smaller, when we have to deal with dilated hearts. I do not deny that these may be fairly successfully overcome in some cases in defining the left border when we have a palpable apex-beat to help us, and also in approximately outlining the base; but they can very rarely be so where the right border is concerned. Its increased distance from the anterior wall of the chest and increased lung covering serve to increase our difficulties. When we take into account all the possible sources of error in such cases, we naturally hesitate about placing much reliance upon cardiac areas as mapped out by percussion. I would not have it inferred that percussion of the heart is useless in all cases. It ought always to be diligently practised, and if every care be taken we may by also taking into account the other methods of examination, in many cases at any rate, arrive at a fairly accurate estimate of the heart's average size; but I feel compelled to deprecate the practice of speaking of the heart as an organ accurately outlinable at all times by percussion.

If this be true, as I believe it to be, it must have an important bearing upon the contention that there is an actual diminution in the size of a dilated heart after a single bath or after a single *séance* of the exercises. There appears generally to be a diminution in the area of cardiac dulness. My own experience fully verifies this, but I have never been able to accept that this was really due to a decrease in the size of the heart. A diminution of 1 to 1½ inches all round—*i.e.*, of 2 to 3 inches in transverse diameter—has been noticed in many cases, and if we try to consider what this means for such an organ as the heart, we find it still harder to admit that this is possible in the space of a comparatively small number of minutes. Dr G. V. Poore has strongly emphasized this difficulty. As the diminution in size is supposed to last for only an hour or so, it seems *à priori* hardly possible that such great differences in the size of a sensitive organ like

the heart could possibly occur within such short intervals of time without giving rise to serious symptoms referable to the heart and brain.

How is the diminution in the area of cardiac dulness to be accounted for if it be not due to a decrease in the size of the heart? The most obvious suggestion is a greater expansion of the lungs, and Dr Poore has already advanced and supported it by observations made upon his own person. I am convinced from my own observations at Nauheim and at home, that the respirations are rendered both fuller and deeper by both the baths and the exercises. This is, indeed, allowed by Dr Bezly Thorne and other authorities. The sense of oppression in the chest experienced by the bather of itself naturally leads to this result, apart from any direct stimulation of the respiratory centre which may be present. As for the exercises, it is to be remembered that they are, with few exceptions, taken in the standing posture, and the increased respiratory action is one of the marked results. The patient has, as a rule, to be cautioned to keep his mouth open and breathe easily. Increased lung expansion is thus brought about, and a greater area of the heart is covered. But if the lung expands anteriorly, why not also in other directions; and it is freely admitted that the lower limits of the lung as determined by percussion remain pretty much the same. Suppose we grant that this line is always pretty accurately determinable and stationary, it seems to me to favour our contention rather than otherwise.

The body is completely immersed in the bath, the head alone remaining above the water, and it naturally follows, from its deeper immersion, that there is more pressure upon the abdomen than upon the thorax. Dr Poore has already pointed this out, and I quite agree with him, and am unable to understand Dr Bowles's contention to the contrary. The abdominal walls are indeed markedly contracted, and the abdominal cavity markedly lessened. I specially noticed this in my first Nauheim bath, and have since found it to be the general rule. This must press the liver upwards, and so constitute an opposing force to the expansion of the lungs downwards. Not only may the fixity of the line of the diaphragm be thus explained, but it may determine a still greater lung expansion in the way of least resistance, viz., anteriorly.

The apex of the lung may also to a certain extent participate; but as expansion is generally much less free in this direction, it is natural that percussion does not usually make out much change here. It may still be argued, however, that the change in the position of the apex-beat yet remains to be accounted for, viz., its displacement inwards and upwards. It is not unusual to find this amount to as much as half an inch or thereabouts, and a full inch has been frequently recorded. It is to be remembered, however, that the apex-beat is not caused by the apex of the heart, but by a part of the anterior wall some little distance from

it, the precise part varying somewhat according to several circumstances. The increased expansion of the left lung may materially affect this, first by covering over a little more of its anterior surface, and secondly by perhaps bodily displacing it inwards towards the middle line. I have tested the influence of the latter factor in the cadaver by inflating the left lung through its bronchus. Moderate expansion displaced the whole heart considerably over towards the right, the apex being the most movable and the base the least. When both lungs were inflated at the same time, the displacement was less marked, but it was always decided, and a very moderate expansion indeed sufficed to effect a displacement inwards of an inch in extent. This displacement was always at the same time upwards, owing to the natural curve of the arch of the diaphragm. On all grounds, then, I fear that we cannot admit that the diminution in the area of cardiac dulness met with in the application of the Schott System corresponds to a decrease in the size of the heart. It is quite possible, nay probable, that some decrease does take place and gradually establishes itself in the course of the treatment, but it is not of daily occurrence, nor is it to be measured accurately by ordinary percussion; and to trust to this as fully as is at present being done is not only to mislead our patients but ourselves, and to shut our eyes to the solution of the problem upon other lines. The evidence derived from auscultation, and occasionally also from inspection and palpation, is more reliable. The heart sounds generally became louder, clearer, and more decided in character. This does not necessarily mean increased cardiac tone, for I doubt if we are able to assert that weak sounds in themselves mean a weak heart, and *vice versa*; and, moreover, loud, clear, and sharp sounds are frequently heard in dilated hearts, and are therefore consistent with an augmentation instead of a diminution of the dilatation. On the whole, however, I think we may say that the heart sounds point to the gradual establishment of an improved condition of heart action. This is still further indicated by the occasional disappearance of a functional systolic or the reappearance of a presystolic mitral murmur. I have only seen one case treated by this method, in which there was visible pulsation in the third, fourth, and fifth interspaces on the left side, close to the sternum. It gradually disappeared after about four weeks; but it ought to have disappeared and reappeared daily for some time, at any rate, if the large temporary decreases in the size of the heart had actually occurred. I notice that Wethered says he saw epigastric pulsation almost disappear after fifteen baths, spread over a period of about twenty days. I do not gather that he observed it after any single application. It is not to be forgotten, too, that apparent spontaneous diminutions in dilated hearts are frequently observed.

*The Pulse.*—The alterations may be estimated by the finger or

sphygmograph, preferably the former. Sphygmograms are useful for recording purposes, and save descriptions, but a too minute reading of them is apt to lead into error. The following agents are all brought into action by the bath, viz., rest, temperature, water, salines, and carbonic acid; and I have endeavoured to test their separate as well as combined effects upon the pulse by trying each in turn.

1. *Rest*.—The patient is enjoined to enter the bath quietly, lie down at once, and remain without moving during the whole period of immersion. Rest is thus secured; and in order to test its effect in a comparable manner I first caused a healthy person to lie quietly upon a couch for fifteen minutes. His pulse diminished in rate and increased in strength. I found this to be even more marked in cases of diseased hearts. Simple changes of posture from the erect to the reclining position, as is well known, are enough in many cases to effect a slight change. The effect of the rest alone is therefore distinct.

2. *Temperature*.—The temperature of the baths varies between 95° and 86° F. An air-bath of 95° F. feels considerably warmer than a water-bath at the same temperature. Something between 85° to 90° F. is probably its equivalent. By means of a tent bed in which the disrobed patient lay, his head being uncovered, I succeeded in testing the effect of an air-bath of about 90° F. The results were rather indefinite. If the temperature rose to about 95° F., as it sometimes did, the pulse was slightly increased in rate, whereas if it remained about 90° F. it was slightly diminished; if it fell a little lower, the pulse diminution became more marked. At or about 90° F. it appeared to be much the same or a little less than was formerly obtained under rest alone, while at 85° or less it seemed to be rather more. This appeared to show that temperatures between 90° and 95° were indifferent, while those under the former figure had a certain definite action.

3. *The simple Thermal Bath*.—In this we have the special mechanical and physical properties of the water added to those of the temperature and rest. The pressure which the weight of the water mass exerts upon the body of the bather is considerable. To the usual atmosphere the weight of the water is added. Mauther says the increase for a column of water 2 feet high is equal to  $\frac{1}{16}$  of the atmospheric pressure, or about 2280 lbs. more than in ordinary air. If we are to judge by the sensations of the patient, a bath of 95° F. feels considerably colder than air at this temperature, the latter being more nearly equivalent to a bath of 105° F. or thereabout. I do not know, however, whether we can say that the temperature effect of a bath at 95° F. is greater than the mere difference between its temperature and that of the body would warrant. The effect of a bath at this temperature continued for fifteen minutes, was usually greater than that of an air-bath 5° lower, or of rest alone. The difference became

more pronounced as the temperature was lowered. At a temperature of 90° F., a diminution of pulse-rate of five to seven beats in a minute, with an increase in strength, was quite common.

4. *The Saline Bath.*—Sodium chloride was next added to the bath in the proportion of 1 to 3 lbs. to the 10 gallons in different cases. Its effect was generally to emphasize the change in the pulse, and to make the bath more agreeable to the patient. This was more noticeable in the stronger baths, which are considerably stronger in salt than sea water. The bath was spoken of as warmer and more stimulating than the simple thermal one. Calcium chloride and barium chloride were next added, first separately and then together, in the proportion of 1½ to 2 ounces to the 10 gallons, without any appreciable effect. Their action without the previous addition of sodium chloride was then tested, with a similar result.

5. *The Carbonic Acid.*—This was mostly added to the artificial baths by means of Sandow's tablets, first recommended to me by Dr Bezly Thorne. Its action was generally quite decided, and led to a further diminution in the rate and increase in the strength of the pulse. When added to a simple plain water-bath its action was very nearly if not as pronounced as when the salines also were present. It added a pleasantness and buoyancy to the bath, very agreeable to the patient, and considerably increased the sensation of warmth. Its presence undoubtedly enables lower temperatures (86° F.) to be more readily borne.

All the factors at work seem to have a certain amount of influence, and the final result may be regarded as due to the summation of their actions. The temperature as administered through the agency of the water is probably one of the most important of these, as its effects are extensive and far-reaching. Temperatures below body-heat applied to the skin cause a contraction of the cutaneous vessels over the part affected, higher temperatures causing dilatation. This result is brought about partly by a direct action upon the walls of the vessels themselves and their local nerve mechanism, and partly by reflex action through the nervous system. Alterations in the calibre of the cutaneous vessels must affect the amount of the lymph stream, and hence the nutrition of the surrounding tissues, and even the flow through the veins and lymphatics. As the whole body is immersed, the area affected is a most extensive one. Further there is reason to believe that the internal vessels are also affected and that extensive cutaneous vascular contraction is always accompanied by a certain amount of internal vascular dilatation, and *vice versa*; and if we are to believe the experiments of Schüller, this is also true of that part of the body not immersed, viz., the head. He observed the vessels of the pia mater in rabbits after trephining the lateral walls of the cranium at both sides of the sagittal suture, and found that cold baths produced dilatation of the vessels

in proportion to the extent of the body immersed, while warm baths had the opposite effect. Winternitz believes that his plethysmograph experiments corroborate these results, and that the contraction produced by thermal influence in a large vascular district causes compensatory dilatation of the vessels in other parts of the body. The plethysmograph registered an increase in volume of the arm during a cold sitz bath, and the opposite during a warm one (95° F.) It will be remembered that this latter temperature is that recommended by the Schott System as most suitable for the commencement of a course of the baths. As it is slightly under the temperature of the skin, we would expect that its thermal influence, if any, would lead to a slight contraction of the cutaneous vessels. Instead of this, a definite dilatation was given, and it is probably to be accounted for by the definite fall in cutaneous temperature caused by disrobing. This would apparently mean a certain contraction of internal vessels, especially of those of the brain. The regular recurrence of the vascular changes may readily enough bring about certain changes in nutrition and tissue metabolism which may come to have an important influence upon abnormal processes. We need not stay to consider the tissue changes, if any, consequent upon abstraction of heat from the body during the bath. The local cutaneous mechanism called into play prevents any reduction in the temperature of the body from such moderate degrees of temperature changes as we employ.

The influence upon sweat secretion must be noticed. During the period of immersion both insensible perspiration and sweat secretion cease. They may, however, be increased afterwards. Whether this has any local action or not it is difficult to say. Heymann and Krebs believe that it saturates the nerve endings and diminishes their excitability. Perhaps it in a measure explains the increased diuresis so frequently observed after the bath. All these actions are more or less independent of the nervous system, and there can be no doubt that there are further actions specially referable to its influence. All thermal influences affect the cutaneous terminations of the ordinary sensory nerves, and thus reflexly the vasomotor and cardio-inhibitory centres. There is no reason to believe that such slight stimuli as our system uses afford any exception to this general physiological law. There will consequently be a slight tendency towards dilatation of the cutaneous vessels of the body generally, and a contraction of those of the head, accompanied by a slight stimulation of the cardio-inhibitory centre, which will tend to increase the strength and slow the beat of the heart.

But we have, further, to consider the influence in this relationship of the various chemical constituents which the bath contains. It is held by many that its saline ingredients act mainly by direct nervous stimulation, and the pleasant warm stimulating sensation

experienced by the patient is pointed to as a proof of this contention. Those of us who have experienced the pleasurable tingling, stimulating sensations which follow a dip in the sea are perhaps too readily inclined to support this. The conditions are far from being similar. The average sea temperature is little over 50° F., and much that we may be inclined to attribute to the salt is really due to the thermal influence. It is easy to see how the sodium, calcium, or barium chlorides, especially the first named, may have some little influence, in virtue of the increase they give to the specific gravity of the water, and the encouragement they may give to exosmotic action. It is mainly, however, a physical or mechanical action, and not a chemical one. The histological structure of the skin makes the possibility of a direct stimulation of the sensory nerve endings very difficult of acceptance.

It is allowed on all hands that no absorption of mineral salts occurs through the skin; but it is maintained by Schott, Keller, and others that a certain imbibition by the skin is possible, whereby the chemical ingredients are brought in contact with the cutaneous nerve endings. Schott supports his view by saying, that when the skin is bathed with plain water after a saline bath, the salt continues to come away for a much longer time than one would expect if it did not penetrate for some distance into the skin. Ranvier's investigations have shown that these nerve endings are not confined to the corium, as was generally believed, but are distributed within the deeper layers of the stratum Malpighii. Even so, however, the mineral salts have a considerable thickness of epidermis to pass before they are reached; and it is difficult to understand how they can penetrate through these layers and yet avoid the lymph channels which lie between the prickle cells external to the nerve endings. Would not an entrance into these lymph channels mean a transference into the lymphatics of the true skin and an absorption into the system which is denied by universal experience? I find it, therefore, difficult to follow Keller's explanations of the observations he carefully made upon himself. He found that a 3 per cent. sodium chloride bath, at 93° F. and of thirteen minutes' duration, has—(1) a distinct diuretic effect, and produces a marked increase of the chlorides of the urine, while plain water baths have the opposite effect; (2) a diminution of phosphoric acid; (3) no appreciable influence upon the nitrogenous waste. Robin, on the other hand, finds that they increase the nitrogenous metabolism and the oxidation of the products of the retrograde metamorphoses of albuminoids. Dr August Schott, in the Laboratory at Breslau, made certain observations upon shaved rabbits. He used a bath with a movable partition dividing it into two chambers, one containing plain water, the other a 10 per cent. salt solution of the same temperature. By raising the partition so as to allow the latter to mix with the

former, he found that the blood-pressure of the animals immersed therein rose 5 to 10 mm. These results have not, so far as I know, been corroborated, and must be received with caution. It is conceivable that the delicate operation of shaving the rabbit—animals which are apt, even under the most favourable circumstances, to prove unsatisfactory subjects for blood-pressure experiments—may have something to do with the result. I cannot say that, so far as the ordinary methods of observation went, my cases disclosed any sign of special stimulation brought about by the presence of the salines beyond the subjective sensations of the patient himself. The increase they produce in the specific gravity and their influence upon osmotic action in connexion with the skin, may indeed be the only influences they call into play. I have endeavoured to estimate their effect upon the vascular system by means of the plethysmograph, using Dr Milne Murray's water manometer as the recording instrument. I wish to record my grateful thanks to him and to Dr Noël Paton for much valuable advice and assistance in this part of my experiments.

So far my observations have not been very definite. They have been made upon two different individuals,—one a lad of 18, and the other an adult male, both healthy. With the patient sitting in an ordinary full-sized bath, water at 95° F. was gradually run in until it entirely covered the legs and pelvis to the level of the anterior superior iliac spines. The plethysmograph recorded practically no change, so that if both these and Winternitz's observations are to be depended on, a different effect would seem to be produced by a rapid and by a slow immersion, to be accounted for probably by the cutaneous mechanism becoming accustomed to the stimulation. The effect of salines and of carbonic acid were tried only in one of the cases. Sea-salt was added in the proportion of 7 lbs. to about 25 gallons of water at a temperature of 90° F. for a duration of several minutes, and gave a negative result, the plethysmograph showing neither rise nor fall. The same result followed upon the addition of 1 lb. of calcium and  $\frac{3}{4}$  of a lb. of barium chloride respectively, over fifteen minutes being allowed for their effects to become established.

So far as these observations go, and they have been too limited to be more than suggestive, they seem to corroborate those made on other lines. The salines would appear to have no direct influence upon the vascular system. It cannot be said, however, that these experiments test in any way their influence upon the nervous system, except, perhaps, in rendering improbable an ordinary stimulation of sensory nerves. If the large tract of cutaneous nerves subjected to the possibility of stimulation by the salines had been affected, might we not have expected some contraction of the vessels of the arm as the result of stimulation of the general vasomotor centres?

On the other hand, if we place any reliance upon the evidence so extensively brought forward by physicians to show that salt-



water baths and douches are of very great value in the treatment of various nervous derangements, we must allow that some form of nervous stimulation would appear to be possible. Whether this may be brought about by electrical contact currents or potentials, which would naturally vary with differently constituted fluids, I am not at present in a position to determine.

The effect of carbonic acid was next tried, the salines being present and the temperature 86° F. A very brisk effervescence was at once obtained by means of Sandow's tablets, and kept up for ten minutes or more. A small and regular diminution in pressure at once showed itself, and continued to increase so long as the record was taken. This showed a contraction of the volume, and thus an increase of pressure in the arm. (The plethysmographic tracings were exhibited.) In Schott's experiments, already referred to, he found that its addition raised the blood-pressure 10, 20, or even 30 mm. Riedlin writes enthusiastically of its influence, and considers that it is the most potent curative agent in mineral water baths. Its presence enables the body to be subjected to much lower temperatures and for longer periods. This he attributes to its stimulating effect upon the skin, by which it is absorbed, when the pressure under which it exists in the bath exceeds that of the CO<sub>2</sub> in the bloodvessels and lymphatics. Schott also supports its absorption under the same circumstances, and believes that its action is mainly due to stimulation of the cardio-inhibitory and other centres. Röhrig, Brémond, and others believe that they succeeded in showing, by experiments on animals, that the skin can absorb gases, and that finely atomised watery solutions of various composition, or such in the form of gas or vapour, will penetrate the skin. If it be absorbed into the blood to any extent, then doubtless it may exert some stimulating influence upon both the cardio-inhibitory and vasomotor centres, judging by the effect of venous blood upon them. This does not follow as a matter of course, as there is the diminution of oxygen to be considered, but I do not know that it is important enough to warrant further discussion here. I can hardly claim to have studied all the experiments made upon the action of CO<sub>2</sub>, but so far as I have done so, I have found no such clear proof as would enable me to maintain that it can be absorbed in the bath by the unbroken skin, nor do I think it necessary in order to explain its physiological effects. We have the bubbles of gas forming an almost continuous covering to the skin, so numerous are they; and though they are constantly being given off and replenished, yet this takes place only to a very limited extent over any given area at one time. The escaping bodily heat may thus be retained for a little by this layer, which will protect the body from the colder surrounding water. This may account in part for the marked feeling of warmth imparted to the skin by the effervescing bath. If the bather moves, the layer of gas is at once liberated over a large area, and a cold sensation is

immediately experienced by actual contact of the skin with the water. One would fancy that, if the feeling of warmth were in any way due to absorption, it would hardly disappear so suddenly on the slightest movement.

Be this as it may, however, there is another and more important action. The bubbles of  $\text{CO}_2$  are constantly impinging upon the skin, and dealing it thousands upon thousands of little blows. The pharynx readily appreciates them every time we take a draught of effervescent water. The cutaneous nerve endings exposed to their action in the bath are actively stimulated. One result of this stimulation is probably a dilatation of the vessels of this area with a contraction of those in the rest of the body, brought about mainly through the influence of the general vasomotor centres. This would explain the rise in blood-pressure recorded by my plethysmographic observations, and presumably also by Schott's experiments. The feeling of warmth experienced over the immersed area may be partly, if not entirely, due to this dilatation of its cutaneous vessels. There may even be other effects, as a slight stimulation of the cardio-inhibitory and respiratory centres; but it cannot, however, be too carefully remembered that just as the stimulus is gentle, so also will its results be gentle, influenced though they may be to a certain extent by the irritability of the individual. The probability, also, of a certain amount of absorption of  $\text{CO}_2$  by the respiratory mucous membrane must not be lost sight of, for the atmosphere which the patient breathes, his head being just above the level of the water, becomes steadily charged with it.

*Effect upon the Heart.*—The primary action upon the heart of stimulation by temperature has long been known, and Röhrig's experiments pointed to reflected impulse of innervation through the vagi as the cause, inasmuch as their division suspended it. Dr Schott believes that the baths act directly upon the heart in the same manner from the triple stimulation of the temperature, salines, and  $\text{CO}_2$ . Dr Douglas Powell would seem to be of the same belief, as in speaking of tachycardia (*Brit. Med. Journal*, Nov. 10, 1894, p. 1039), he says—"And doubtless, as in the very analogous cases of exophthalmic goitre, the strong brine baths of Nauheim, by their stimulating effect on the vagus centres, will prove of value." Now, so far as the evidence before us goes, we are led to think that the baths have very little influence indeed in this direction, and that, such as it is, it is attributable slightly to the temperature, chiefly to the  $\text{CO}_2$ , and but little, if at all, to the salines. The diminution in rate and increase in strength of the cardiac contractions produced in this way must be very slight indeed, and altogether inadequate to immediately bring about any gross change in the size of the heart.

*Effect upon the Respiration.*—It becomes more active. It diminishes slightly in rate, but becomes markedly deeper, and it

is to be remembered that deeper breathing is much more effectual than more frequent breathing in carrying off the products of a more active internal respiration. An increased elimination of  $\text{CO}_2$  and moisture and an increased absorption of oxygen will thus be brought about. This will, no doubt, directly influence tissue metabolism and nutrition, but may also increase the ordinary influences of respiration upon the circulation, by the more oxygenated state of the blood affecting the vasomotor centre, by its influence upon the heart and bloodvessels, brought about by nervous action, and by its direct mechanical effect upon blood-pressure. Thus deeper inspirations promote the return of the blood-flow through the lungs to the heart, and deep expirations have an opposite effect. I doubt if this can have any appreciable influence, but the compression of the abdominal viscera ought to facilitate the return of blood to the heart, and so stimulate it by increasing its endocardial pressure.

*Effect upon the Blood.*—Winternitz and his assistants found that cold baths produced an increase of the red blood-corpuscles, and also of the white, with a 14 per cent. increase of the hæmoglobin. The maximum effect was sometimes manifested an hour afterwards, and lasted often for two hours. Warm baths had the opposite effect. He does not regard this as due to new formation, but to increased activity of the heart and circulation and improved nerve tone, the red and white cells being thus driven out of the organs in which they are accumulated. Kellogg obtained corroborative results, and agrees with his explanation. Grawitz agrees with the results but not with the explanation. He believes that warm baths cause the blood to be less concentrated, acting by a stimulation of the vasomotor nerve apparatus, leading to a contraction of the vessels, and preventing transudation of fluid into the tissues. So far as I have been able myself to try this method, I have obtained very little difference indeed from temperatures between  $95^\circ$  and  $85^\circ$  F.,—a trifling increase, but nothing more.

It would, therefore, appear that "the System" acts mainly by regularly repeated alterations in the blood-pressure, the distribution of the blood, the osmotic actions, and the various external and internal secretions. The natural ebb and flow between cutaneous and internal vessels is brought more systematically and regularly into play. Such changes may undoubtedly stimulate tissue metabolism and lead to the correction of vices and the establishment of improvements in general nutrition. The heart will participate in these improvements, and it is well known that its beat varies largely in consequence of changes in its own metabolism. There is no evidence whatever to support claims which maintain a great and direct influence upon the heart, whether by nervous channels or by greatly lessening the peripheral resistance. There is, indeed, every reason to believe that although the cutaneous vessels may be somewhat dilated, the general sum of arterial

pressure, and hence the peripheral resistance, is actually somewhat increased. This seems to be entirely in keeping with the improvement observed in the cardiac conditions, for among the physical or mechanical circumstances of the heart which affect its beat, the most important is perhaps the amount of distension of its cavities. The contractions of cardiac muscles, like those of any other muscle, are increased by the resistance, within certain limits, which they have to overcome. A full ventricle will contract more vigorously than one less distended, while if the limit at which resistance is beneficial be passed, an over-distended ventricle will fail to contract at all.

There is yet one other influence which must not be lost sight of in any summation of the different methods of action of "the System," and it is that of the mental factor, to which, however, I feel loth to refer, as we have no means of properly explaining or estimating it. The suffering lay or even medical mind is markedly susceptible to the influences of any line of treatment which can apparently offer such striking and rapid results by means so tangible, pleasant, and easily understood. A patient convinced of his daily improvement, and of his certain recovery a few weeks later, is often already half cured. Much faith is a wonderful healer, and when it is accompanied by a "complete change" and pleasant society, such as it is the duty of a health-resort to provide, it may work wonders, and we must be on our guard lest we give our treatment the entire credit of results which it may have had very little hand in producing.

It will be necessary to say but a few words further in special examination of the resistance exercises. The physiological explanation underlying their action is simpler than that of the baths, to which they may form valuable adjuncts. They may, indeed, at times prove useful where the baths are contraindicated, but, on the whole, they are inferior to them both in range of applicability and in efficacy. They are sensible and well thought out exercises, excellently suited to accomplish their purpose of bringing each group of muscles in the trunk and limbs into action in turn. They substitute regular and graduated movements capable of being perfectly controlled by the skilled attendant, so as to suit a considerable variety of conditions for exercise often irregular, spasmodic, and ill-regulated which the patient, left to his own devices, is apt to indulge in. When thus employed so as not to overtax the patient at any one time, their physiological action resembles that of all other beneficial muscular exercise. There will be dilatation of the bloodvessels within the muscles, increased tissue metabolism, and more rapid elimination of effete materials. This will gradually bring about a better general nutrition. I cannot see how even the most gentle of such exercises can fail to press upon the veins and favour the return of the venous blood to the heart. Dr J. F. II. Broadbent thinks that this failure may occur.

It is an ingenious suggestion put forward to account for the diminution apparently observed in the size of the heart, which is thus due to a lessening of peripheral resistance afforded by this means together with the vascular dilatation within the muscles. But if we deny this direct diminution in the size of the heart,—as, for the reasons already given, I think we must do,—the suggestion becomes unnecessary, as also does the even more hypothetical and improbable one of Dr Schott, viz., reflected vagal stimulation brought about by *afferent impulses* carried to the cardio-inhibitory centre by means of the *motor nerves*. We know of nothing which leads us to place such a difference between gentle and vigorous muscular exercise. They must both hasten the circulation and favour, although in different degrees, the return of the venous blood to the heart. This will be most marked in the abdominal blood-vessels. Compression of the abdomen instinctively results with the employment of the resistance exercises. In this way an increased intracardial stimulation follows, and will naturally lead to an increased blood-pressure. It is possible that the vascular dilatation within the muscles is not compensated for by a corresponding contraction in bloodvessels in other regions, and is, besides, greater in amount than that of the increase due to the quickened venous flow. Some observations of Dr Lauder Brunton in his Harveian Oration of last year seem to support this. There would in this case be an actual lessening of peripheral resistance and a diminution in blood-pressure, but this is rendered improbable by the decrease in rate and increase in strength of the pulse.

The physiological problems raised by "the System" are both many and complex, and there must be much still hidden from us which the future may reveal, and which may bring about a more perfect understanding of its actions. So far as they are at present known to us, it is readily seen that its scope of application is far wider than that of any individual drug, but to assert its applicability to all classes of cardiac derangements, reserving only advanced arterio-capillary sclerosis, aneurisms, and serious myocarditis, is surely in so many words to proclaim its impotence. It is but natural that it should fail. Evil habits of nutrition which measure their existence by months or years are not to be got rid of by one or even two courses of "the System" of a few weeks' duration at Nauheim or anywhere else. I have already met with failures and recurrences such as the present literature of the subject makes no mention of, and they have but served to increase my belief in its value when used in suitable cases. While we remember that it is capable of doing much good, we must not forget that it is also capable of doing harm. It has now been sufficiently proved to warrant its trial in suitable cases, but they must be chosen with judgment. To use it rashly and with too great expectations is certain to lead to disappointment and to reflect injuriously upon "the System" itself. Moreover, as at present

enunciated it does not seem to me to be as useful as it might. It is at once too wide and too restricted,—too wide in its claims and too restricted in its limitations. Why, for instance, should we pause at 86° F. ? Why not go further in suitable cases, and make use of the well-known effects of still lower temperatures, inasmuch as we can so easily regulate their influence by the duration of time we employ them. The resistance exercises are good so far as they go, but why should they not be combined with massage, or passive or active exercises of different kinds? Eccles, Campbell, Lauder Brunton, and others have testified to the value of the latter. An intelligent use of dumb-bells and other gymnastic appliances ought to prove quite as effectual in some, if not in all, cases as the resistance exercises themselves, and they have indeed done so in my hands in the opportunities I have so far had of putting them into practice.

I have herein endeavoured to lay before you the lines of a sound physiological working of "the System," which promises to prove a valuable remedy in many cases of chronic heart disease, but whether I have succeeded or not, I am not without hope that I have prepared the way for the clearer understanding and appreciation of its applicability and usefulness.

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#### BIBLIOGRAPHY.

1. BENEKE.—"Zur therapie des Gelenkrheumatismus und der damit verbundenen Krankheiten," 1872.
2. AUG. SCHOTT.—"Die Wirkung der Bäder aufs Herz," *Berliner klinische Wochenschrift*, 1880, No. 25, ss. 357 to 359; and No. 26, ss. 372 to 374.
3. GROEDEL.—"Pneumatometrische Beobachtungen über den Einfluss verschiedener Bäder auf die Respiration," *Berliner klinische Wochenschrift*, 1880, No. 22, ss. 314 to 315.
4. GROEDEL.—"Zur Behandlung Herzkranker," *Berliner klinische Wochenschrift*, 1883, No. 25, ss. 381 to 384.
5. THEOD. SCHOTT.—"Beitrag zur tonisirenden Wirkung Kohlensäurehaltiger Thermalsoolbäder aufs Herz," *Berliner klinische Wochenschrift*, 1883, No. 28, ss. 428 to 431.
6. AUG. SCHOTT.—"Beiträge zur physikalischen Diagnostik des Herzens," *Centralblatt für die medicinischen Wissenschaften*, 1881, ss. 419, 436, 449, 467.
7. AUG. SCHOTT.—"Zur therapie der chronischen Herzkrankheiten," *Berliner klinische Wochenschrift*, 1885, No. 33; ss. 424 to 527; No. 34, ss. 545 to 548; No. 35, ss. 559 to 562; No. 36, ss. 582 to 584.
8. AUG. SCHOTT.—"Die Bedeutung der Gymnastik für Diagnose, Prognose, und Therapie der Herzkrankheiten," *Zeitschrift für Therapie*, 1885.

9. THEOD. SCHOTT.—*Die Behandlung der chronischen Herzkrankheiten.* A separate Pamphlet published by Grosser, Berlin, 1887.
10. THEOD. SCHOTT.—“Zur Pathologie und Therapie der Angina pectoris,” *Deutsche medicinische Zeitung*, 1888.
11. THEOD. SCHOTT.—“Zur acuten Ueberanstrengung des Herzens und deren Behandlung,” *Verhandlungen des Congresses für innere Medicin, Neunter Congress*, 1890, ss. 448 to 477.
12. THEOD. SCHOTT.—“Herz und Herzkrankheiten,” *Real. Encyclopädie der gesammten Heilkunde*, xxii. 1890, ss. 5 to 35.
13. THEOD. SCHOTT.—“The Treatment of Chronic Diseases of the Heart by means of Baths and Gymnastics,” *The Lancet*, 1891, vol. i. p. 1143.
14. THEOD. SCHOTT.—“The Mineral Waters of Nauheim: their Action, Uses, and Effects.” London, 1894.
15. THEOD. SCHOTT.—“Zur Behandlung des Fettherzens,” *Deutsche medicinische Wochenschrift*, 1894, xx. s. 561.
16. THEOD. SCHOTT.—“Zur aetiologie der chronischen Herzkrankheiten,” *Wein. med. Presse*, 1892, ss. 33, 1145.
17. GROEDEL.—“Bad Nauheim und die Behandlung der chronischen Herzkrankheiten,” *St Petersburg Medicin. Wochensh.*, 1893, F. x., ss. 141 to 151. Do. do., *The Lancet*, March 30, 1895.
18. MOELLER.—*Die Traitement des Maladies du Cœur par la méthode des Drs Schott de Nauheim*, deuxième édit., 1893, published by A. Manceaux, Bruxelles.
19. ISRAËL (Copenhagen).—*Om Nauheimkur, balneologisk-gymnastik Behandlung efter Schott*, 1891.
20. BEZLY THORNE.—“The Treatment of Chronic Diseases of the Heart by Baths and Exercises, according to the method of Dr Schott,” *The Lancet*, 1894, vol. i. p. 1117.
21. BEZLY THORNE.—“The Treatment of Chronic Affections of the Heart by Baths and Exercises,” *British Medical Journal*, 1895, vol. i. p. 524.
22. BEZLY THORNE.—*The Schott Methods of the Treatment of Chronic Diseases of the Heart, with an account of the Nauheim Baths and of the Therapeutic Exercises.* Churchill, London, 1895.
23. STURGE.—“Note on the Treatment of Dilated Heart as practised at Nauheim,” *Brit. Med. Journ.*, 1895, vol. i. p. 527.
24. BABCOCK.—“The Schott Method of Treating Chronic Diseases of the Heart by Baths and Gymnastics,” *The Journal of the American Medical Association*, November 11, 1893.
25. WETHERED, F. J.—“The Treatment of Chronic Diseases of the Heart by Baths and Gymnastics as practised at Nauheim,” *British Medical Journal*, 1894, vol. ii. p. 1045.
26. ARMSTRONG, W.—“The Nauheim Treatment of Chronic

- Cardiac and allied Diseases," *Liverpool Medical Journal*, July 1895.
27. SMYLY, Sir P. C.—"On the Treatment of Enlarged Heart by certain movements, as taught by Dr Th. Schott of Bad Nauheim," *Dublin Journal of Medical Science*, Sept. 1894.
  28. BROADBENT, J. F. H.—"On the Treatment of Chronic Heart Disease by the Methods of Dr Schott of Nauheim," *The Practitioner*, 1895, p. 385.
  29. BROADBENT, Sir WILLIAM.—"Some Remarks on the Mode of Action of the Treatment and its Indications," *The Practitioner*, 1895, p. 394.
  30. GREENE, RICHARD.—"The Schott Treatment for Chronic Heart Diseases," *The Hospital*, October 19, 1895.
  31. SAUNDBY.—"Remarks on the Nauheim (Schott) Treatment of Heart Disease," *Brit. Med. Journ.*, Nov. 2, 1895, p. 1081.
  32. LESLIE THORNE-THORNE.—"Cases of Heart Disease treated by the Schott Method," *The Lancet*, Jan. 4, 1896, p. 26.
  33. POORE.—"The Schott Treatment of Heart Disease:" Letter, *British Medical Journal*, 1895, vol. ii. p. 1195.
  34. BUTLER, HARRIS, and THEODORE FISCHER have letters on the subject in *British Medical Journal*, Nov. 16, 1895, p. 1262; and ROBERT L. BOWLES, BRADLEY, and HARRY CAMPBELL also have letters in the number of Nov. 23.
  35. CLAUDE WILSON.—"Saline Baths in Graves' Disease," *The Practitioner*, September 1895.
  36. HEITLER.—*Die Percussionsverhältnisse am Normalen Herzen*. Published by Hölder, Wien, 1891.
  37. SCHÜLLER.—"Experimentalstudien über die Veränderungen der Gehirngefäße unter dem Einflusse äusserer Wasser-applicationen," *Deutsche Archives für klin. Med.*, Bd. xiv.
  38. WINTERNITZ.—*Ueber thermische Wirkungen auf die Blutvertheilung in Die Hydrotherapie*, Bd. i. p. 115, 1890.
  39. BARUCH.—*New York Medical Journal*, Nov. 30, 1890.
  40. KELLER, H.—*Correspondenz Blatt für Schweizer Aertze*, Basel, April 15, 1891.
  41. ROBIN, A.—*Bulletin de l'Acad. de méd. de Paris*, May 19, 1891.
  42. RIEDLIN.—*Hygeia*, Stockholm, Sept. 1891.
  43. WINTERNITZ.—*Blätter für klinische hydrotherapie und verwandte Heilmethoden*, Vienna, November 1893.
  44. KELLOGG.—*Modern Medical and Bacteriological World*, Battle Creek, Michigan, December 1893.
  45. GRAWITZ.—*Centralb. für klin. Med.*, Leipzig, Jan. 13, 1894.
  46. HEYMAN and KREBS.—"Untersuchungen über die Wirkung der lauwarmen Fluss- und verschiedener anderer Bäder," *Virchow's Archiv*, Bd. l. Heft 1.
  47. MAUTHNER.—*Die Heilkraft des kalten Wasserstrahles*, Wien, 1837.



48. RÖHRIG.—*Die Physiologie der Haut Experimentell und kritisch bearbeitet*, 1878.
49. RÖHRIG.—“Expériences physiologiques sur l’absorption cutanée,” *Comptes rendus*, lxxiv.
50. LAUDER BRUNTON.—*The Harveian Oration*, London, 1894; also “On the Use of Rest and Massage in Cardiac Affections,” *The Practitioner*, vol. li. No. 3, 1893; “On the Effects of the Kneading of Muscles upon the Circulation, Local and General,” *Journal of Physiology*, vol. xvii. No. 5, 1894; “Atheroma and some of its Consequences, with their Treatment,” *The Lancet*, Oct. 12, 1895.
51. SYMONS ECCLES.—“Mechano-Therapy in Chronic Diseases of the Heart,” *The Practitioner*, vol. liii. p. 106, Aug. 1874.
52. CAMPBELL, H.—“The Mechanical Treatment of Heart Disease,” *British Medical Journal*, 1894, vol. ii. p. 1101.

The following have appeared since the delivery of this paper, whilst it was in the press:—

53. HERSCHELL.—“Critical Remarks upon the Nauheim Treatment of Heart Disease,” *The Lancet*, Feb. 15, p. 413, 1896. He failed to get any favourable results, and entirely condemns the System.
54. BROWNE, RALPH.—“The Nauheim Treatment of Heart Disease”: a Letter, *The Lancet*, Feb. 29, 1896, p. 586. He emphasises the influence of the CO<sub>2</sub> and its absorption by the lungs.
55. BEZLY THORNE.—“Notes on Certain Changes in the Cardio-Vascular System which are induced by treatment according to the Schott Methods,” *The Brit. Med. Journ.*, March 14, 1896, p. 653. He distinguishes an oval, bipartite, and conoid form of dilated heart as made out by auscultatory percussion, which is in my opinion quite untrustworthy, and has given him illusory results.
56. KINGSCOTE.—“Fifteen Months’ Practice of the Schott Methods for the Treatment of Chronic Affections of the Heart,” *The Lancet*, March 21, 1896, p. 761. He gives seven cases, but I fail to follow him in his *rationale*.
57. BEZLY THORNE.—“Self-poisoning in Heart Disease, and its relation to the Schott Method of Treatment,” *The Lancet*, March 21, 1896, p. 755. An Address delivered to the Leamington Medical Society. He refers to catarrhs of the gastro-intestinal tract, and the probability of auto-intoxications arising therefrom poisoning the blood, and thus reacting upon the heart.
58. BOWLES.—“The Nauheim and Schott Treatment of Diseases of the Heart”: A paper read before the Harveian Society of London, March 19, *The Lancet*, March 28, 1896, p. 850. He supported the method strongly, and gave four corroborative cases.

59. Sir WILLIAM BROADBENT.—“Note on Auscultatory Percussion and the Schott Treatment of Heart Disease,” *The Brit. Med. Journ.*, March 28, 1896, p. 769. He objects, as Herschell and I have done, to the method of auscultatory percussion, and strongly supports the contentions brought forward by me in this paper, viz., the untrustworthiness of percussion alone, the limitations which ought to be placed upon the use of the System, the harm which its injudicious use may bring about, and the necessity of selecting carefully only suitable cases.
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*The President* said that they were very much obliged to Dr Leith for having brought before them a method of treatment that appeared to have attracted attention recently, and also that he had employed a certain amount of the healthy scepticism with which all new methods of treatment should be regarded until they had been analysed and their effect ascertained.

*Dr George Balfour*, in response to an invitation from the President, said that he so entirely agreed with every word Dr Leith had said that he had nothing more to add. He thought Dr Leith was perfectly justified in pointing out that the limits of cardiac dulness varied indefinitely every few seconds, and to base any fixed diagnosis of the size of the heart upon these percussion limits was to reason from a fact which had no real foundation whatever. He thought Dr Leith had very clearly expounded the various methods with which the System of Schott was concerned, and he also agreed with him that it was not at all a system of treatment that could be employed with impunity in all cases. In a certain number of carefully selected cases it was of great advantage; but there was no doubt that to a great extent its influence was upon the minds of patients quite as much as upon their bodies, and especially upon their hearts; while, if employed rashly and in cases that were unsuitable, it was certain to be unavailing, and might be even disastrous.

*Dr Gibson* said that Dr Leith's paper was of such value that even although one agreed entirely with the observations he had brought before them, and had therefore but little to criticise, one ought to offer him a meed of praise, not merely for the courage with which he had brought forward his views, but also for the great care he had spent on the matter. He would be inclined, however, to cross swords with Dr Leith as to the accuracy of the modes of ascertaining the size of the heart. The only way in which they could prove their methods was necessarily in the cadaver. Over and over again he had been able to demonstrate that they could map out the size of the heart to within  $\frac{1}{16}$  inch all round without very much difficulty. He thought Dr Leith would substantiate him in making that statement. There was greater difficulty, no doubt, on the right side, but even then the resources at hand for accurate percussion led them to surprisingly correct results. As

to changes in size, no doubt, as Dr Balfour admitted, the heart changed its size every  $\frac{1}{100}$  second, one might say. The changes in size from systole to diastole were perhaps surpassed by those produced by respiration. When forced inspiration could produce 60 mm. of negative, and forced expiration 90 to 100 mm. of positive, pressure, it was very obvious that the changes in the arterial and venous pressure, and consequent changes of pressure in ventricles and auricles, must vary within very great limits indeed; and these changes in inspiration and expiration he thought went a long way to explain what they observed as the result of passive exercises. Possibly in the near future they might see with their own eyes what went on there. He had been observing in Prof. Grainger Stewart's wards the results of passive exercises and resistance exercises on the size of the heart; and, speaking generally, he would only say this, that mere massage seemed to have little influence on the size of the heart, while resistance exercises had considerable power of modifying its size, but he thought that came about through changes in respiration. With regard to the remedial effects of all these measures, he thought they could not expect very much from them. Patients came back from them in a state of high exhilaration, but it did not take much in the shape of a catarrh, gastric or respiratory, to upset the results. In some of the patients in the Royal Infirmary who had undergone resistance exercises, what benefit was produced was nearly evanescent.

*Dr James Ritchie* agreed with Dr Gibson that *in the cadaver* it was possible by percussion to ascertain, with a considerable degree of precision, the size of the heart. The theories supported by Dr Leith were in accordance with the teaching of the text-books, but Dr Ritchie thought that there must be some fallacy in the experiments on which this was based. It seemed to him to be contrary to reason that a dilated heart, which was unable satisfactorily to carry on its work, the action of which was rendered more irregular and its rate increased by exertion, physical or mental, or by reflex causes, that such a heart could be benefited, its action rendered slower and more regular, by an increase of pressure within the vascular system; that is to say, by increasing its work. A heart which was only slightly enfeebled might be so benefited, through the increase of tension improving the nutrition of its walls. He believed that the baths at Nauheim and the passive exercise produced beneficial results by diminishing the peripheral resistance to the circulation by means of a dilatation of the capillaries. He understood Dr Leith to say that saline baths had no stimulating effect. Was it not a matter of everyday experience that sponging with tepid or cool salt water was more stimulating than fresh water at the same temperature? It was a most useful method of treatment during recovery from debilitating ailments. He believed that the baths at Nauheim acted not only by dilating the capil-

laries, and so diminishing obstruction to the outflow of blood, but also by stimulating the heart through the vagus, and so producing a more perfect contraction of the heart muscle.

*Dr Leith*, in reply, said that he must first of all state that he had listened to Dr George Balfour with a great deal of pleasure, and also, in a sense, with some disappointment, because he was hoping that he would perhaps find out some source of error that he (*Dr Leith*) had not discovered. He paid the greatest deference to what he said on the subject, and it was very gratifying to him to find so great an authority as Dr Balfour entirely agreeing with him, especially upon such an important subject as that of heart percussion. He confessed that his own opinion, as he went on with these observations, underwent marked changes. He was not then in the healthy position with regard to percussion that he was in now, and which he hoped his friend Dr Gibson would some day reach. He had percussed the cadaver perhaps as often as Dr Gibson or Dr Ritchie, and he agreed with them, and had indeed stated it that night, that in almost every case such percussion was correct except in cases of marked emphysema or where the right lung largely covered the heart, and he was quite sure that in these cases even Dr Gibson's or Dr Ritchie's practised percussion would fail. But what he wished especially to emphasize was that the difficulties in the way of accurate cardiac percussion were in some way peculiar to life, and did not exist in the cadaver. Estimation of the heart's dulness during life could only at best be approximate. Dr Gibson had referred to the influence of respiration on the pressure on the left side of the heart. He (*Dr Leith*) doubted if it could increase endocardial stimulation of the left side of heart very much. Experiment had not, at any rate, been able to determine it. Apart, however, from the slight differences of opinion which might exist between some of the speakers and himself on minor points, he was gratified to learn that they supported him in the restrictions he had put upon the System, and this was all the more agreeable seeing that this was the first time that any adverse criticism had been made public. (*Dr Herschell's* paper has since then appeared, *The Lancet*, Feb. 15.)

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#### Meeting V.—February 5, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. ELECTION OF MEMBERS.

THE following gentlemen were elected Ordinary Members of the Society:—J. Cormack Smith, M.B.C.M., 9 Brunton Place, and R. D. Clarkson, M.B.C.M., 20 Graham's Road, Falkirk.

## II. EXHIBITION OF PATIENT.

*Mr Cotterill* showed a patient who exhibited a well-marked example of a disease which was not very common, and which was of considerable importance, in so far as it was apt to lead to something more serious later on in a number of cases,—leucoplakia, also called ichthyosis of the tongue, or superficial glossitis. Its importance lay in the fact that in many cases it was followed by epithelioma,—not, it appeared, by any direct transformation, but from its being very often associated with fissures and cracks which might take on malignant character. As to the pathology of the condition, it was thought by most authorities to be due to a combination of tobacco and syphilis. He had not been able to elicit any syphilitic history in this case. There was ulceration in the middle of the tongue. When there was a specific history, one might expect a somewhat better result from iodide. He had been giving it for three or four days. The usual treatment was by iodide of potash and local mercurial lotions, bicarbonate of soda to clean away the epithelium, and painting with chromic acid and things of that kind. But it was admitted on all hands that the treatment was very inefficacious. It was of importance to the general practitioner to recognise this condition, because *Mr Cotterill* could not help thinking it would have a very important bearing on the question of life insurance. There were many grades of the disease less severe than that seen in this patient. The case was under treatment, and in a few weeks *Mr Cotterill* might have an opportunity of showing him again.

## III. EXHIBITION OF SPECIMEN.

*Mr Cotterill* showed an EPITHELIOMA OF THE LARYNX which he had removed in hospital that day. The patient had been sent to *Dr McBride*, who reported that the right cord was immobile. *Mr Cotterill* did a preliminary tracheotomy, and put in a large tracheotomy-tube a few days ago, and went on to the second half of the operation that day. On splitting the thyroid he found the vocal cords healthy, though the right cord was fixed, but on passing his finger up towards the mouth he felt on one side a considerable amount of thickening. He split the parts right up to the chin, dividing the hyoid bone in the middle line, and separating the soft parts at the back of the larynx, and pulling them up as well as he could from the pharyngeal wall, turned aside the sterno-hyoid and sterno-thyroid muscles, got to the back of the disease after a somewhat deep dissection, and got it all removed, as far as he could tell. A large part of the pharyngeal wall had to be removed along with it.

## IV. EXHIBITION OF PHOTOGRAPHS.

*Dr Dawson Turner* showed, by means of the lantern, a series of PHOTOGRAPHS taken by the RÖNTGEN PROCESS. He said that he

never knew whether he was going to have a successful negative or not. He might take one photograph, and it would come out pretty well, and then take another, when he would sometimes get no effect at all upon the negative. He was entirely in the dark at present as to what the rays were which affected the negative plate, how they were to be concentrated, or best directed on the plate. From the experiments he had made he was led to the belief that the rays could be reflected, contrary to the supposition of some, who held that they could not be reflected, polarised, or deflected by the negative, etc., and therefore were not rays of light. He had found the rays act only at very short distances. If the plate were more than a yard, or even  $1\frac{1}{2}$  foot or so from the vacuum tube, there was no effect. An ordinary exposure was thirty minutes. He could not understand how some could take photographs in four minutes. He had taken one that day in a quarter of an hour, and it was not very well marked. The thickest part of the body he had so far been able to go through was the wrist. This negative showed pretty well the radius, ulna, and some of the carpal bones. The cuneiform was apparently not in its expected position. He had shown the photograph to Sir William Turner, who at once remarked on the absence or displacement of the cuneiform. If the process could be applied to the thicker parts, no doubt many points of anatomy would be cleared up, such as the position of the patella in flexions of the knee-joint. The successful photographs of human anatomy he had as yet taken were those of foot, hand, and wrist. He would now show several photographs on the screen,—first one of the foot, and one of the hand, in which the skeleton came out distinctly. The interarticular intervals showed well the transparency of the articular cartilage to the rays. The medullary cavity of some of the bones could also be seen in a fainter tint. Thickenings of the plantar epidermis came out darker than the surrounding parts. The terminal phalanx of the little toe, they would observe, was turned directly inwards, and he was inclined to regard that as its normal position. He next showed a photograph of a pencil-case and half-opened knife. These had simply been placed against the wooden shutter of the camera, through which the rays passed easily. Next a purse with two metallic clasps, an outer and an inner, containing a florin and key. The metallic structures came out dark, the rest of the purse hardly appearing at all; next three cigars in an aluminium cigar-case. The cigars, each with its paper band, came out clearly, and even the manner in which they were rolled was in some degree visible. There was also a faint dark zone indicating the elastic band with which he had fastened the case to the shutter. He next showed a photograph of a lady's hand with ring on finger; also one of the wrist in position of supination, showing the styloid process of the ulna lying dorsal, and even a little external; next one of coins in a

purse ; next of a key, pen-nib, and various other articles ; also one of a frog, taken by Mr Campbell Swinton of London, in which the limb bones came out distinctly. He had himself taken one of a fish, in which the stomach, owing evidently to its containing something solid, had come out distinctly. He also showed one of a razor in its case, taken by Mr Swinton.

*The President* said that they were all obliged to Dr Dawson Turner for his demonstration of this new invention, which might become important in the near future, since it might enable them to see parts in the interior of the body, and obtain distinct traces of foreign objects there, being more or less of an endoscope. It might give them a more accurate and clearer idea of the position of parts in the interior of the body.

## V. ORIGINAL COMMUNICATIONS.

1. *Dr Byrom Bramwell* brought before the Society a remarkable case of CALCAREOUS DEGENERATION OF THE HEART AND ARTERIES, with rapidly-developed symmetrical subcutaneous tumours in the axillæ, elbows, groins, natal folds, and popliteal spaces, occurring in a young man, aged 25, affected with advanced cirrhosis of the kidney, the right having been completely destroyed fourteen years previously by a pyelo-nephritis ; with a pathological report by Dr Lovell Gulland. Dr Bramwell stated that this was perhaps the most remarkable case which had come under his notice during the twenty-seven years that he had been in practice. The patient was unmarried, aged 25, and was seen with Dr Menzies on April 16, 1894. He complained of extreme debility, loss of flesh, and the presence of symmetrical swellings in the axillæ, groins, inguinal folds, and popliteal spaces. When 11 years of age, he had had a severe illness, and had been confined to bed for six months. After recovering, he enjoyed good health until he went to America, eighteen months before his death. In America he was exposed to great hardships, and as the result completely broke down and was laid up for six weeks. During this illness he lost a stone and a half in weight, and became very weak and pale. In December 1892 he came back to Edinburgh and remained for a year with his friends. During this time he did not make any special complaint, and did not consult a doctor. About the middle of January 1894 he bought an engineer's business in Glasgow, and for some weeks subsequently he went through much hard physical work, fitting up his workshop and lifting heavy machinery. He stated that during this time he did not feel anything amiss. During the last week of March 1894 he strained his back in lifting a heavy piece of machinery. He came to Edinburgh two days later, and consulted Dr Menzies, who, after a careful examination, was unable to detect any evidence either of external or internal disease. A week later the patient experienced pains

in the muscles of the thighs and legs, which were thought to be rheumatic in character. A few days after this he directed attention to the condition of the axillæ, groins, gluteal folds, and popliteal spaces, which, on examination, were found to be swollen, hard, and brawny. In the course of the next week or ten days the debility and anæmia rapidly increased and the subcutaneous swellings enlarged. The urine contained a trace of albumen and a few pus corpuscles. When Dr Bramwell saw him on April 16, 1894, he was extremely feeble, much emaciated, and markedly anæmic. The skin of the face, arms, and abdomen was of a dingy brown colour, resembling the condition in an advanced stage of Addison's disease. The temperature ranged from 90° to 100°, the pulse from 100 to 110; it was extremely small and weak. The heart's action was exceedingly feeble, the impulse imperceptible. The red blood-corpuscles were diminished, 2,700,000 per cubic millimetre. The urine was copious, of low specific gravity, and contained a small quantity of albumen; it was loaded with phosphates. No tube casts were detected. Hard brawny swellings were present in each axilla, the flexor aspect of each elbow, over the anterior superior spinous process of the iliac bones, in the posterior folds of each axilla, and in the popliteal spaces. The condition of the skin and subcutaneous tissues at the seat of the swellings more closely resembled a cancerous or sarcomatous infiltration than any other condition which had come under Dr Bramwell's notice. The swellings appeared to be infiltrated with some hard (calcareous) material, and there were many small hard subcutaneous nodules about the size of a small shot in the adjacent skin. On sitting up the patient complained of giddiness and a feeling of faintness. On May 1, 1894, when he was again seen in consultation, the subcutaneous swellings were considerably enlarged, and both radial arteries were now found to be absolutely rigid and pulseless. The femorals and popliteal arteries were in a similar condition. On May 5 pericarditis developed, and the patient died on May 7. After death the radial, iliac, femoral, popliteal, and some other of the peripheral arteries were found to be entirely calcareous, and converted into rigid tubes. The aorta and cerebral arteries were perfectly normal. The heart was considerably enlarged, weighing 1 lb. 2 ozs., and was a splendid example of calcareous degeneration. The valves were quite healthy. The right kidney was completely destroyed by old disease, and converted into a thin firm mass of fibroid tissue. The left kidney was in a very advanced state of cirrhosis. The microscopical examination, made by Dr G. Lovell Gulland, showed that the chief pathological changes were present in the heart, in some of the arteries, in the kidneys, in the subcutaneous tumours, and in certain parts of the skin. There were patches of calcareous degeneration throughout the wall of the heart, the individual muscular fibres being calcified. Many of the arteries were com-



pletely calcified. The arteries in the subcutaneous tumours were calcified, and the surrounding tissue infiltrated with calcareous deposits. In the heart, and still more in the subcutaneous tumours, the connective tissue elements surrounding the calcareous deposits were in a condition of proliferation. Putting all the facts of the case together, Dr Bramwell concluded that the following was in all probability the sequence of events:—(1) That at the age of 11 the patient suffered from pyelo-nephritis of the right kidney,—that the right kidney had at this time been entirely destroyed; (2) that the exposure and hardships to which he had been subjected in America eighteen months before his death had induced disease in the other kidney, or more probably had aggravated a cirrhotic process which was already present in the left kidney, and that had slowly and gradually progressed until the extreme condition of cirrhosis which was found after death had been produced; (3) that, for some reason or other, which the author could not explain, the blood had become impregnated with calcareous salts in solution; (4) that owing to the advanced cirrhotic condition of the kidney, the calcareous material had (instead of being excreted by the kidney) been retained in the blood and finally deposited in the walls of the heart and peripheral arteries, and extravasated into the subcutaneous tissues; and (5) that local irritation, the result of mechanical movement, probably played a part in determining the deposit of calcareous material in the walls of the heart and in the subcutaneous tissues over the joint surfaces, gluteal and axillary folds, etc. The localization and symmetrical distribution of the skin and subcutaneous lesions could only, Dr Bramwell thought, be accounted for by supposing that some mechanical condition or conditions (such as movement and stretching) was concerned in their production. The absence of any symptoms of Bright's disease and uræmia was, considering the condition of the kidneys, very remarkable. A photograph and water-colour drawing showing the appearance of the subcutaneous tumours, the subcutaneous tumours themselves, the heart, the arteries, the kidneys, and a series of microscopic sections and drawings illustrative of the minute pathology of the case, were exhibited at the meeting.

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*The President* said they would be glad to hear any remarks of members present on this very curious and interesting case that had been so vividly depicted by Dr Bramwell.

*Dr Russell*, in response to the President's invitation, said that he was sure they had all followed with deepest interest the record of this extraordinary case. He no more than Dr Bramwell had ever seen a case at all like it. Of course it had bearings on other cases. Extensive calcareous degeneration and infiltration occurring along with kidney lesion was met with from time to time in aged persons. He had himself, like Dr Bramwell, met with this exten-

sive calcareous affection of arteries, with accompanying advanced renal cirrhosis, but always in old people. The special feature in this case was its occurrence at this very early period of life. As to the possibility of such occurrence, he had a case at the Infirmary "out patients" last Tuesday, a man of 33, with abdominal aneurism, and very markedly thickened and somewhat calcareous vessels. He had been operated on some years previously for aneurism of the popliteal artery. He had recovered, and now turned up with aneurism of the abdominal aorta, and this very marked condition of his arteries. That, of course, was the kind of case that was recalled to one by this of Dr Bramwell's. He knew that a calcareous condition of the heart did occur, but he had not met with any case of such extensive incrustation as evidently existed here, extending even to the endocardium of the left ventricle. He had not heard of anything at all approaching such a condition as that. The condition of the skin was also very curious, and he thought that Dr Bramwell's interpretation of the changes there was correct. The appearances to the naked eye were evidently those of a fibrous hyperplasia, which were exceedingly suggestive of a malignant process affecting the cutis vera, extending into the subcutaneous tissues and surrounding individual fat lobules. One could make out with the naked eye in various parts of this fibrous tissue areas of calcification which, he presumed, originated, as Dr Bramwell had suggested, around individual vessels. Whether the extensive fibrosis was really due to calcareous infiltration or not was a point which might be questioned, and yet it was exceedingly difficult to account for it on any other hypothesis. That in the main Dr Bramwell's conclusions were supported by the facts he thought none of them could doubt, and that the incompetence produced in the kidney led to this extraordinary accumulation of lime salts which were deposited in the arteries and also at certain parts in the subcutaneous tissues, leading in the latter to thickening and tumour-like growths. As one of the physicians present, he fancied he might assure Dr Bramwell that they were, as a Society, very much indebted to him for bringing before them a case of such very great interest, a case which he thought would prove practically unique when put on record.

*Dr Young* said that he could not but associate this case in his mind with a case which he once saw, in which not the arteries but the muscles were affected with calcareous deposition in early life,—he referred to myositis ossificans. He had mentioned the case to the Society when a paper was read by Prof. Annandale on wry-neck. It was that of a little girl of 6 or 7 years. He only saw her once, and did not take her address, so that he was unable to show her to the Society. It was an extreme case of what Rindfleisch had called myositis ossificans; in fact, the sternomastoids were quite rigid calcareous rods, and the pectorals were

also affected. It was a good many years since he had seen her. He cited the case in illustration of the tendency, even in early life, to the deposit of calcareous matter.

*Dr Foulis* said that he rose to direct attention to a very important matter that had been partly alluded to by Dr Bramwell,—the connexion between kidney disease and the deposition of lime salts. No doubt, as Dr Russell had already emphasized, they saw calcareous degeneration very frequently in old people, and it was extremely common to find, in this cirrhotic degeneration of the kidney, advanced calcareous degeneration of the arteries, and perhaps of the rings surrounding the pulmonary and aortic orifices of the heart. He could recall a case in which Dr Bramwell performed the post-mortem, and found most marked calcareous degeneration of the large bloodvessels, aortic valves, and rings at orifices of heart in an old lady. In recent times he had frequently seen cases of marked calcareous degeneration in old people, but invariably most marked in those cases in which kidney degeneration was most prominent, and in these cases the prominent pathological feature was the extremely arenal condition of the individual. It would be well to emphasize this as probably accounting for the general calcareous condition, *i.e.*, the failure of the essential organ of elimination. He thought Dr Bramwell had shown that this man was practically an arenal individual; and how he could have lived for so many years without showing symptoms he could not understand.

*Dr Armour* said he would just like to ask Dr Bramwell if he could explain why in this case the man did not present symptoms of the absence of excretion of urea? Such kidneys as these suggested to one that they had hardly any function whatever.

*Dr Bramwell*, in reply, said that he was very much obliged to Dr Robertson and Dr Russell for their remarks. With regard to the question of urea, the man was passing three pints of urine daily, and he must have been excreting much more urea than they would think possible. That was just one of the points of clinical interest in the case that it was so difficult to explain. He could give no explanation, could merely state the fact that it occurred, and that there were no symptoms of uræmic poisoning. As to the interesting condition referred to by Dr Young, he had never had an opportunity of seeing it, but no doubt it had a relation to the present case. Of course, as Dr Foulis had said, one saw the condition in advanced life; but in some that he had seen there was no evidence of cirrhotic kidney at all. When he first went into practice he saw an old gentleman with every artery in the body calcareous, who lived for many years. He remembered Dr George Murray of Newcastle remarking, on seeing the patient, that he ought to be bottled as a whole and put in a museum. That was one of the cases without any marked cirrhotic condition.

2. *Dr Lockhart Gillespie* said that he did not intend to read his paper, on THE WEATHER, INFLUENZA, AND DISEASE, FROM THE INFIRMARY RECORDS, in full, but would call the Society's attention to one or two of its main points. He had divided it into four parts, the first of which dealt simply with the data on which he based his conclusions. He had taken the last seven years' admissions into the medical wards, numbering 27,569 cases. He had divided these according to the system affected, and also according to the number of admissions during each week for the different years. He first investigated the admissions of the different classes of disease in connexion with weather. With regard to weather, he had looked into, more particularly, two of the elements—the element of type, *i.e.*, whether it was cyclonic or anticyclonic; and, instead of taking the mean temperature, since he did not think health was affected so much by mean temperature as by the variations, he had taken the extremes in each week, *i.e.*, weeks in which temperature did not rise above 60°, and weeks in which it did. This divided the year very fairly. With regard to the type, he found that during the seven years, for 30·5 weeks in each year the prevalent type was cyclonic, for 21·5 anticyclonic. The warm weeks were to cold as 29 to 22, roughly. The first thing he noticed with regard to the type was that for three of these seven years cyclonic weather was markedly in excess. The years he had taken were from 1st October 1888 to 1st October 1895, and it was in the second, fourth, and sixth of these years that the cyclonic element was most marked. Respiratory cases were admitted in larger numbers during these three years, and the weekly admissions of respiratory cases during the cyclonic weather were much in excess of the admissions during the weeks in which anticyclonic conditions prevailed, while in the other four years the exact opposite occurred, *viz.*, respiratory cases smaller in total number, and also smaller in proportion to total admissions during the cyclonic weeks, compared with those admitted during the anticyclonic. He could not find the same relation between the temperatures. He had tables, which he would pass round, showing some of the points. It was especially noteworthy that while respiratory cases were most abundant during cyclonic weather and when the maximum temperature was below 60°, cases of pneumonia were admitted in much greater number during anticyclonic weather, and were only slightly increased when the temperature fell below 60°—*i.e.*, pneumonia, being an infective disease, was not so much affected by the temperature as ordinary respiratory cases. Pleurisy corresponded with other respiratory disorders. Cardiac disease was more rife in cyclonic and in warm weather; kidney disease also in cyclonic weather. Rheumatism underwent a very great increase indeed in anticyclonic weather, and also when the temperature was low. The others he hardly needed to mention, except that the digestive system was more affected in anticyclonic weather, when the barometer was high

and the weather dry. The tables explained themselves. On going over these figures he found that the incidence of influenza during the years in which he worked at the records altered the results so markedly that he had included a note on influenza, which had rather exceeded in size the original work. He showed a chart which contained a considerable number of entries. The seven years were shown, each divided into weeks. He had indicated by a red line the absolute number of respiratory cases admitted each week. He had also taken the average of every five successive weeks, and by noting it on the chart had obtained a smoothed curve. Below were the actual number of cases admitted each week into the medical wards with influenza. It was at once apparent that there had been an epidemic in the winter of 1889-90, a small epidemic in the spring of 1891, a great epidemic in the winter of 1891-92, a very small epidemic in the spring of 1893, a considerable epidemic in the winter of 1893-94, a well-marked epidemic in the spring of 1895—*i.e.*, six epidemics in six years; and if they counted up the weeks, they found sixty-eight weeks in six years, or one in every four and a half. He had, by the kindness of Dr Douglas, obtained a record of the number of cases of influenza from the Post Office, and the Post Office curves corresponded with the Infirmary curves. In the first epidemic the percentage of postmen affected was 6.5 per cent.; in the second, 3.5 per cent.; in the third attack, 13.9 per cent.; in the fourth, 1.5 per cent.; in the fifth, 11.6 per cent.; and 7.4 per cent. in the last epidemic—*i.e.*, a mean percentage of 7.9 of the whole Post Office in the six attacks. Another line on the chart showed the weekly type of weather, red for cyclonic, blue for anticyclonic. The next line indicated temperature, blue for under 60°, red for over 60°. Cardiac cases were also “Bloxomed” by fives, as were rheumatic and nervous cases. The total admissions were also “Bloxomed” to get a smoothed-out curve. After an epidemic of influenza one noted an increase of cardiac cases two or three months after the attack. He showed a small diagram in which each of the lines indicated the average normal percentage in relation to the total admissions into the medical wards. Red indicated the percentage below and above normal during each attack, and blue the percentage above or below normal to the total admissions after each attack. Cardiac and nervous cases were above the average after each attack of influenza. With regard to weather conditions, the normal was thirty cyclonic weeks to twenty-two anticyclonic annually; but if they took the conditions that occurred before, during, and after the epidemics, they found the following striking facts brought out:—He had taken the six weeks preceding each of these epidemics, thirty-six weeks in all. Taking the normal mean for these, they found cyclonic twenty-six, anticyclonic ten. For these weeks, however, the actual figures were, cyclonic thirty-one, anticyclonic five, *i.e.*, an immense increase of cyclonic weather before each attack of influenza. The first two

weeks of each attack, or twelve weeks in all, were three cyclonic to nine anticyclonic in type, while the normal average for these weeks was actually the opposite, nine to three. Influenza was preceded by cyclonic weather, and commenced in dry anticyclonic weather. Before each attack they found the rainfall above normal and temperature below normal. In the first two weeks of the epidemics the rainfall was small, almost *nil*, and the temperature also low; but during the whole sixty-eight weeks of the epidemics the temperature had been normal, and the rainfall practically normal. For the four weeks following the epidemics the rainfall was above normal, *i.e.*, the six epidemics in Edinburgh had been preceded by wet stormy weather of predominantly cyclonic type, had begun in dry, cold weather of predominantly anticyclonic type, had continued during normal weather, and had subsided in weather with temperature rather above the normal for the periods in the seven years, and the rainfall very much above normal. He had many more facts which he would not trouble them with. He had taken statistics of the last four epidemics that occurred some years ago in the Infirmary. From the Infirmary records he found clear evidence of an epidemic of influenza in the winter of 1848-49, in February to May 1851, in January to April 1855, in October to February 1857-58. The charts which he had made out would show how influenza affected the Infirmary then. The most extraordinary epidemic of all was that of 1848-49, when the number of cases rose in one week to 39 per cent. of the total admissions, excluding fever cases, for which there was not then a special hospital. There was no very marked increase in the respiratory cases admitted. Out of the whole ten epidemics it was only in that of 1848-49 that the respiratory cases were not increased. A large number of digestive cases were admitted during the course of the epidemic. He regretted that owing to the late hour he could not go more fully into the subject at present.

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*The President* said that they had all listened with great interest to Dr Gillespie's paper. It was a very wide subject which he had tackled,—*viz.*, the interaction between climatic conditions and disease. They trusted this was only a sample of the results which he would get from such investigations, more particularly if he could throw light on the causation of influenza, that extremely obscure and many-sided disease. He had referred in his paper to the epidemic of 1848-49. He (Dr Robertson) could bring quite clearly to his recollection that epidemic, and the consternation that prevailed in Edinburgh at that time in consequence of the occurrence of that unknown disease. The term influenza was then brought prominently forward, and the disease was viewed with great alarm by all the inhabitants of the town.

*Dr Craig* said that he would just like to ask one question. Had they conclusive evidence that the epidemic of 1848-49 was pre-

cisely of the same kind as the disease with which they were more familiar as having occurred during recent years, because the name influenza was rather widely used at one time.

*Dr W. T. Black* said that this was a very interesting subject. Having had some experience in hygiene, he had taken great interest in this paper. It seemed to have involved a great deal of work, collecting statistics, drawing conclusions, diagrams, and averages, and had occupied time quite out of proportion to the space given it that evening. Without entering into the details of the various diagrams and tables, he might state that from the hygienic or sanitary point of view it was less important than from the meteorological point of view. They could all recognise that the human frame was made for a variety of climates; that man travels from the north to the south pole through various climates both on sea and land without great deterioration of constitution; further, that the barometer may be high or low, and the altitude might vary from Alpine resorts to the seaside, yet the constitution tolerated them equally well. But they must discriminate between weather and climate. The seaman came through cyclones, typhoons, and the tropics, and was none the worse. If they regarded bad weather as injurious to the human frame, how could they get a reduction of mortality in town or country? If they could not improve the weather, they could not lessen the mortality. The same weather had endured in England for 1000 years, but they had reduced the mortality considerably—from 30 to 40 per thousand in cities to 15 or 20—so that there was something beyond weather that produced disease. There were the various germs, bacilli, and effluvia floating about in the atmosphere, coming and going according to laws not yet perfectly understood. The meteorological view of disease might afford useful employment to those interested in it, but it did not bear so much on the hygienic aspect of the case as was generally supposed. There was one point on which he congratulated *Dr Gillespie*, viz., on his having made a good name for anticyclonic weather. During the present year they had had a great deal of it, and should therefore all be in a healthy state. He congratulated them on the fact that this weather would probably continue, and would enable them to get through the rest of the winter with improved health, and be ready for the excursions and festivities of spring.

*Dr Gillespie*, in reply, said that he had two questions to answer. *Dr Craig* had asked as to the type of disease in 1848. It was rather extraordinary that if they took the percentage of respiratory cases to the total admissions, it worked out exactly the same in the earlier as in the later epidemics, that figure being very much over the normal for the whole period. The pneumonia percentage during these attacks was exactly the same as during the last series; and so on, all the way through, they found that its effect upon disease in the four previous epidemics had been almost

identically the same as in the later. He did not say that the weather caused the influenza; he merely said that it facilitated its spread. If influenza was present, it got a start when the weather changed; and once it had started in epidemic form, it did not matter what sort of weather came on afterwards. It might be hot or cold, the epidemic went on until there was a plentiful downpour of rain.

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### Meeting VI.—February 19, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. EXHIBITION OF PATIENTS.

1. *Dr James* showed—(a.) A patient who, he said, had come in from the country three or four weeks ago. The history was that he had possibly received an injury to the left foot five months ago. This, however, was doubtful, and could not have been very severe. Shortly afterwards he became lame in the left leg, and had severe pain in the bones of the left foot. He was treated outside for this with rest and fomentations, without effect. Then he was sent in to the surgical department of the Infirmary. On examination there was found some thickening about the left foot, but its nature was not very clear. The point of medical importance was that in examining the leg they found distinct wasting of the muscles of the left leg, and to a certain extent also of the left thigh. He walked very much better than when he came in, but the foot was slightly everted, and movement tended to bring on pain. With wasting of the muscles of the left leg and foot, there was marked increase in reflexes. In both legs, especially the left, the knee-jerk was very much increased. Ankle-jerks also increased, especially the left. Knee clonus and ankle clonus present on both sides, especially the left. There was no doubt some thickening about the left tarsus; but the point seemed to be that, as the result of some peripheral source of irritation, his lumbar cord was in a state of increased irritability, causing increase of knee and ankle jerks, and clonus. They thought it a case of functional increase of irritability of lower part of cord. But in such cases the muscles were ordinarily well nourished, while in this case there was some wasting of muscle. There was no reaction of degeneration, so that he thought the wasting was simply the result of disuse. He thought, therefore, that this was a case of increased irritability of the lumbar cord from irritation proceeding from the bones of the foot. Since the patient's admission they had given him baths, on the theory that it might be rheumatic. After each bath the mobility was increased.



(b.) A boy, *æ*t. 9 years, with PSEUDO-HYPERTROPHIC PARALYSIS, the hypertrophy well marked in the muscles of the back. He had the ordinary symptoms. There was no family history. The hypertrophied muscles were found to be stronger proportionally as a whole, and reacted more to electricity than the muscles weakened and at the same time atrophied. There was nothing very special about the case.

(c.) A case of HYDRONEPHROSIS. The patient was a man, *æ*t. 46, who was admitted with a distinct fluctuating swelling in the left lumbar region. There was a history of a swelling of exactly the same kind in 1878, from which he had recovered, and he remained well until it returned some months ago. They had not been able to make out the cause. There was no history of calculus. But when comparatively young he had had what he called an attack of dysentery. He seemed to have been ill then for several weeks, passing mucus and blood; and in connexion with the hydronephrosis, they thought it possible that there was some inflammation about the sigmoid flexure which, as the result of cicatricial contraction, had obstructed the passage of urine down to the bladder. They treated him with rest in bed for a few days. The hydronephrosis did not diminish. They accordingly punctured, and took off about 20 ozs. of dilute urine with some blood. The sac collapsed, and was found to communicate with the ureter—the urine becoming bloody. As the sac filled with water the ureter again became obstructed. They soon saw that the amount of coloration in the urine diminished, and the sac enlarged again. They therefore aspirated a second time, and took care to empty it completely. After this there was blood for a day or two, but now there was no appearance of the kidney enlarging, and the urine was perfectly clear. The sac was collapsed, and he was safe for some time. The last tapping was done a week ago on Sunday. In drawing it off they felt carefully for a stone, but could not make it out. The obstruction was therefore probably due to some band pressing on the ureter.

2. *Dr Logan Turner* showed a woman, *æ*t. 57, who had been sent to him by *Dr R. A. Fleming*, suffering from LARYNGEAL PHTHISIS, on December 24, 1895. At that date the lungs showed consolidation of both apices in front and behind, with numerous coarse and fine crepitations. The sputum was copious, and contained many tubercle bacilli. The larynx showed swelling of arytenoid cartilages, especially the left; and the left ary-epiglottic fold was prominent, and tending to the pear shape. The true vocal cords were congested, but no ulceration could be detected. On the posterior wall in the inter-arytenoid space there was a well-marked raised uneven edge indicating ulceration below. She could only speak in a hoarse whisper, and suffered from dysphonia, dysphagia, and sometimes considerable dyspnoea. Treatment

consisted in bi-weekly intra-laryngeal applications of the mixture recommended by Dr Chappell of New York—Creasote, ʒjss.; ol. ricini, ʒiij.; ol. gualther., ʒiij.; ol. hydrocarb, ʒj.; menthol, gr. 10. Improvement began to show itself in a fortnight, and on February 19, 1896, the accompaniments in the lungs had disappeared, except a few at the right apex behind. The sputum was much diminished. The swelling of arytenoids and left ary-epiglottic fold had disappeared, the vocal cords were but faintly reddened, and the ulcer on the posterior wall was healed. The voice was strong; there was no dysphonia, dysphagia, or dyspnœa; and the general condition had improved. During the last fortnight she had been taking guaiacol carbonate, 5 grs. thrice daily, with much benefit to the cough.

3. *Mr Cotterill* showed a case which, he said, was of considerable interest, chiefly perhaps in connexion with the question of proper treatment. The case was one of PLEXIFORM ANGIOMA, sometimes called CIRROID ANEURISM; but the latter term was generally restricted to a tortuous condition of the arteries, the former to those in which the veins were also involved. The vessels mostly at fault in this case were the transverse facial, temporal, posterior auricular, and to some extent the occipital. The tumour began, as many of these cases did, with a traumatism. He got a blow with a cricket ball when 18 years of age, which resulted in a black eye, with a good deal of swelling, which became organised, and did not subside as it should have done. He had a photograph of the condition at 18, when the swelling was restricted to the eye, but from that time it spread enormously outwards, backwards, and downwards. On lifting the eyelid one found the eye at the bottom of a deep pit, in what he believed to be a normal condition, except for slight conjunctivitis. He could not feel the facial artery on the affected side, although there was a groove in the bone, just where it ought to be. The patient had been under treatment more or less for the last thirty years, but nothing had been done for some years past. He had been treated in Britain by various well-known surgeons, including Prof. Spence, but nothing had done any good. In front of the ear, one could feel a very large vessel, the transverse facial, enormously enlarged, and the posterior auricular, not so much enlarged, could be felt distinctly pulsating. The branches of the temporal were very much enlarged. Above the eye the bone had been encroached upon, as was sometimes the case. Whether this was periosteal thickening or eating of the tumour into the bone, it was not easy to say with certainty. It might be a question of interest whether this tumour was restricted to the branches of the external carotid, or communicated through palpebral or supraorbital branches with the internal carotid. In the latter case treatment would be more difficult. The tumour had been growing fast lately, especially

downward. Perhaps also it tended to drag from its own weight. On its surface there was a large cicatrix, due to the fact that at one time he took the advice of an unqualified practitioner of the female sex, who applied some caustic, causing ulceration,—so far interesting that it did not kill him. The ulceration healed, and that encouraged one as to future proceedings. As regarded treatment, one could, in the case of a small angioma, either excise or pass subcutaneous ligatures through it, much on the same principle as in ligature of a pile; but neither of these plans could be thought of in this case. Ligature of the separate vessels feeding such a tumour had not been found successful. It was said that not a single instance of the kind had resulted in cure. Ligature, however, of the vessels at a distance—*e.g.*, external carotid—had in a very few cases been followed by cure. In one or two cases both external carotids had been tied, and in one case at anyrate a cure resulted. Thirty per cent. of such cases dealt with by ligature of the common carotid had proved fatal. In the present case ligature of the branches would not be satisfactory, for there must be dozens of them. Probably ligature of external carotid, together, possibly, with electrolysis, would be best. These two methods combined might help to solidify part of the tumour, and something might be done subsequently in the way of excision. There were some hard lumps here and there which might be the result of experiments in galvano puncture done several years ago. Pulsation in the tumour was never very strong. After compressing the main vessel so as to hinder blood supply, and then squeezing the blood out of the tumour, it filled very quickly,—not so quickly as when the common carotid was free, but still quickly enough to show that they could not trust to ligature of the common carotid to prevent hæmorrhage. He would be glad to have the opinion of the Society on the question of treatment. These tumours occasionally occurred on the body and sometimes on the limbs, but usually on the scalp. There was one form of treatment of which he thought the patient in this case would probably not approve, *viz.*, amputation. It was suitable when the tumour occurred on the foot. In reply to a question by the President, Mr Cotterill said that there was a very slight bruit.

4. *Mr Caird* showed a boy who had been suffering for two years and seven months from HIP-JOINT DISEASE. He had diarrhœa and was emaciated. Obviously there was nothing to be done but amputation. This was done after the usual circular method, and he went back in rather a collapsed condition. About 3 A.M. he found it necessary to transfuse, and injected about 12 ozs. of saline solution. The patient had since been on a voyage up the Baltic, and had returned stronger than before. He got along wonderfully well on one leg.

## II. EXHIBITION OF SPECIMENS, ETC.

1. *Dr Johnston* related the clinical history of a case of DEPRESSED FRACTURE OF THE SKULL, of which the specimen was shown. The patient was a seaman, at. 56 years. In July 1889, while working on board ship in Burntisland harbour, he fell from a yard to the deck, a height of 12 feet, was picked up unconscious, and taken to a lodging-house in the town, where he was attended by a medical man. He was unconscious for three days. After that he regained consciousness, and in a few weeks resumed work. There was, he said, a depressed fracture of the skull in left temporal region. The patient lived six years after that, for five of which he followed his occupation. He had no symptoms beyond giddiness, while walking along a plank. The depression was almost circular, about 3 centimetres in diameter, almost like a trephine wound. Some years after the accident he began to suffer from epithelioma of the tongue, from which he died in about twelve months.

2. *Mr Cathcart* showed—(a.) The specimens from *Dr Johnston's* case. Being anxious, he said, to bring out the feature as well as possible, he had cut away all the facial bones and hardened the brain *in situ*. When the brain was thoroughly hardened he carefully removed the skull-cap, and they could now observe that the skull-cap showed, besides the depression in the frontal region, another depression of no less extent in the occipital region, unsuspected during life. In order to see the effect produced he had made a gelatine cast of the interior of the skull-cap, showing the great depression in the frontal and occipital regions. The brain itself showed marked depressions, although from the contour being so much broken up by the natural convolutions, they were not so marked as in the skull. The dura mater was adherent to the skull, but not to the arachnoid or pia. The brain was quite healthy below. There was simply depression without inflammation, softening, or any other change. This case appeared to be an interesting confirmation of the view that it was not so much depression of the surface that caused the mischief as irritation produced by sharp spicules or fragments. The frontal region could probably stand more without showing symptoms than most other parts of the cranial contents.

(b.) A specimen of SARCOMA OF THE LUNG, sent by *Mr Alexis Thomson*. The patient had been admitted to Deaconess Hospital. *Dr Cattanaeh* would relate the clinical history.

(c.) A plaster cast of RIGHT ARM and a DISSECTION OF THE ELBOW, showing backward dislocation of ulna and radius. The capitellum was very much atrophied, and the head of the radius rounded. All the movements were impaired.

(d.) A specimen of ELEPHANTIASIS OF LABIUM MAJUS, which had

been sent to him from Mombasa by Dr Macdonald. He himself possessed a specimen of twice the size.

(e.) A specimen of HYDRONEPHROSIS WITH A VALVULAR CONDITION OF URETER, as in Dr James's case. The special point was the development of plain muscular fibre, not only in the wall of the sac, but also in the form of a series of bands stretching across it. Both to naked eye and microscopically there was distinct evidence of their muscular structure.

(f.) He showed for *Dr Felkin* a GROOVED CATHETER, *i.e.*, with the lumen replaced by a deep groove. It could thus be more easily kept clean. The grooved form might be useful in cases of retention from spasm, but would not be suitable for the small catheter required in cases of stricture.

3. *Dr Cattanaeh*, in the absence of *Mr Alexis Thomson*, related the history of a case of SARCOMA, of which the specimen of the lung was shown. The patient was a miner, aged 24, who had come to hospital in June 1895. There was a small swelling of about the size of a walnut underneath the skin near the internal malleolus. It was diagnosed a neuroma, from the pain and position. Mr Thomson cut down and removed a mass of about the size of a small hen's egg near the internal malleolus, and above that a similar mass of the size of a hazel nut. Microscopic examination showed it to be a sarcoma. The wound had healed perfectly in two weeks. Two months later there was recurrence at the seat of the wound, and he came to hospital again. Mr Turner performed amputation, and also cut down and removed a gland in the groin. The patient recovered and went to Convalescent House, but in November he came back with a larger mass of the size of a bantam's egg in the groin. Mr Thomson cut down and removed the mass with some difficulty. There was considerable venous hæmorrhage, and he had to ligature the femoral vein. About six days afterwards the patient showed lung symptoms, brought up some blood, and his temperature went up to between 105° and 106°. They thought of pulmonary embolism. By and by there were signs of pleuritic effusion. On three occasions they aspirated and took away blood-stained fluid, about 50 ozs. each time. He suffered great distress after the tappings, became very dyspnoic, and died two days afterwards. At the post-mortem there were found sarcomata under the skin, on the scalp, and on opening the chest both lungs were found studded all over as specimen shown. There was nothing in kidney or spleen, but all the other organs, even the heart muscle, had sarcomatous growths. There was no thrombosis at seat of operation.

4. *Dr Byrom Bramwell* showed—(a.) Some water-colour drawings and photographs of a typical case of ANÆSTHETIC LEPROSY which had come under his observation a few years ago. (b.) Water-colour drawings of a case of CANCER OF THE LIVER, which presented

some features of clinical interest. (c.) Photographs and a series of drawings representing a GLIOMATOUS TUMOUR, which was limited to the right side of the pons varolii, and which produced unilateral paralysis of a large number of cranial nerves.

5. *Dr Gillespie* showed some specimens of ALBUMOSES GOT FROM A SINGLE SPECIMEN OF URINE—one of the most extraordinary urines he had ever seen. The patient passed a large quantity of urine, and there was also a history of albuminuria. In January last, out of 2860 c.c. he got about three times as much albumose as albumen. There was a trace of albumen and various albumoses. He had under observation a lady with albuminuric retinitis and albumosuria, so that the absence of albumen from the urine did not signify the absence of Bright's disease. He had seen it in other cases in which there was no history of renal disease.

6. *Dr Leith* showed—(1) a CASE OF PHLEGMONOUS GASTRITIS—naked eye and microscopic specimens. He said—John M'K., æt. 49, clay-pipe maker, a strong healthy man, felt somewhat out of sorts on Thursday, Jan. 9, but continued at work all that day and the next. He remained in bed during part of Saturday, getting up for a few hours only. The bowels moved freely during the day. He took a good breakfast early on Sunday morning, but two hours afterwards vomited, and this continued at intervals for two or three hours. He slept well that night, but was wakened about 5.30 A.M. on Monday by severe pain in the region of the stomach. Vomiting began shortly afterwards, and continued throughout the day. It was at first clear, but soon became bile-stained. *Dr William Wood* (to whose kindness I am indebted for these early notes) saw him for the first time at 11.30 P.M. He had an anxious look, and was perspiring slightly. His pupils were contracted, and he was restless, with slight delirium. The tongue was dry and covered with a dirty white fur. Temperature  $101^{\circ}$ ; pulse 110, full and somewhat tense. He complained of slight frontal headache and severe pain over the region of the stomach, which was aggravated by the lightest percussion. *Dr Wood* saw him five times between 1 P.M. on Tuesday and 1 A.M. on Wednesday, and his condition did not improve. Hydrocyanic acid, bismuth, liq. morph. hydroch., and ice, were given internally, and poultices, linseed and mustard, were applied over the stomach, and relieved the vomiting. On Tuesday his temperature was  $102^{\circ}\cdot5$ , and pulse 86. He was quite conscious, and complained of much thirst, and marked pain in the region of the stomach, and also in the right hypochondriac region. The bowels had not again moved, and a soap and turpentine enema was given, and repeated three or four times, with no effect. There was tenderness on pressure in the left hypochondriac region, but no tympanitis until after 1 A.M. on Wednesday morning. He was admitted on the forenoon of that day, Jan. 15, to the Royal Infirmary, under the care of Mr

Duncan. Peritonitis was suspected, and Mr Thomson performed laparotomy very shortly after his admission. A general peritonitis was found, showing some concentration towards the right iliac region; but the vermiform appendix was found to be normal. No cause of the peritonitis could be ascertained, and although the patient rallied for a time after the operation, he died late in the same day. At the sectio I found an early acute peritonitis, showing only a very little whitish lymph here and there, and no noticeable concentration towards the right side. A small white patch about the size of the thumb nail was observed over the lesser curvature of the stomach, about  $1\frac{1}{2}$  inch above the pylorus. This proved to be purulent lymph, about  $\frac{1}{8}$  inch thick, adherent to the peritoneal coat and lesser curvature of the stomach. The peritonitis had spread from it. The stomach wall was felt to be greatly thickened, especially in the region of the pylorus. The cavity contained about 2 ozs. of pale yellow fluid. The mucous membrane was pale and greatly swollen throughout, especially in the region of the pylorus, where it showed a slight excoriation of its upper layers. It had a peculiar pulpy feel, but there was no visible ulceration. Sections showed the mucous membrane and muscular coats to be clearly defined and separated from one another by a greatly thickened submucosa of a yellowish-white colour. It was exactly the colour of pus, but no fluid exuded. It was almost half an inch thick from the pylorus upwards for three or four inches. Thence it spread upwards in varying thicknesses to the cardiac end (well seen in specimen). No break in either the mucous or the muscular layers could be found at the seat of the purulent lymph on the peritoneal surface. The duodenum was healthy, and the bile flowed freely into it. All the other organs were healthy.

*Microscopic Appearances.*—The submucous coat is nearly everywhere infiltrated with small round cells, some with a well-stained single round nucleus, others in which the nucleus stains badly. A few of the cells are larger, with a bigger, sometimes lobed nucleus. The amount of cellular infiltration varies greatly in different parts; in some no other structure is to be seen, and the cells form a mass of great thickness (Microscope 4). In other parts they form a layer many times thinner and are less abundant, allowing of the thin reticulum of the normal submucous coat to be seen. Its vessels are dilated (Microscope 1.) The muscularis mucosæ appears normal, but is broken at places by strands of similar small round cells. These are well seen where small vessels penetrate it from the submucous into the mucous coat. The mucous coat itself is greatly infiltrated with these cells. They form dense, thick strands here and there, surrounding and separating the gastric glands (Microscope 2). In other parts they are absent. They have apparently spread from below. The gastric glands themselves are not much altered (Microscope 1). Here and there on the free surface of the mucous

membrane there is a network of considerable thickness of fine fibrin threads whose meshes are filled with round, badly-staining cells (Microscope 2). The muscular coats show the same infiltration, both in masses passing outwards, especially along the lines of the vessels, and sometimes in strands passing horizontally between the muscular bundles, and separating them from one another. Many of these cells are large, with distinct clear protoplasm, and a large well-stained nucleus (Microscope 3). Many of the cells reach the peritoneal coat, and spread themselves out over it, mingled with fibrin threads. In the submucous coat there are immense numbers of micrococci, mostly arranged in well-formed chains, many single, many diplococci, and here and there are dense staphylococci-like masses. These were present in the exudation on the mucous coat, very sparsely in the superficial part of the mucous coat, in enormous numbers in the submucous coat, especially in its deeper part, and in places in the muscular coat, wherever the exudation was present, and generally all over the peritoneal coat. Many of them are free, and many enclosed within leucocytes (Microscope 5). This is a beautiful example of the diffuse form of idiopathic phlegmonous gastritis, an extremely rare disease. The other form, the localized, forming a distinct abscess, is still more rare. It is met with as a primary condition, as in this case, or as a metastatic condition in a general pyæmia. Diffuse phlegmonous gastritis is more common in males than females, in the proportion of about four or five to one. Thus of 41 cases which I have been able to collect, 33 were male and 8 female. Its ætiology is extremely obscure, and alcohol, injury, and dietetic errors have been blamed, but are obviously insufficient. In the present case the patient had not been addicted to alcohol. The cocci were most probably the cause, but there was nothing to explain their appearance or method of entrance. Prognosis: Practically always fatal in from five to twelve or fourteen days. The circumscribed form may be much longer, and is a little more hopeful.

(2.) HÆMORRHAGIC PANCREATITIS (Dr Russell's case)—naked-eye and microscopic specimens. There was much thickening in the neighbourhood of the pancreas, causing the organ to appear considerably enlarged. Shining through this, it appeared to be of a slate or reddish-brown colour. The peritoneum covering it was somewhat thickened and separated from the anterior surface of the gland by a fatty-looking tissue. This is quite unusual, as even in very obese bodies the peritoneum is closely approximated to the anterior surface of the gland. The layers of the transverse mesocolon appeared to be normal, and showed no hæmorrhage; nor was there any hæmorrhage retro-peritoneally in its neighbourhood. No areas of fat necrosis in the omentum, peritoneum, or abdominal fat were observed. The thickening around the pancreas was chiefly above and below it, and was seen to consist chiefly of fatty tissue,



with bands of fibrous tissue running through it. Here and there also, close to the pancreas, were small pea-like foci, sometimes a little larger, filled with a granular putty-like material, like dried-up abscesses. These were mostly outside the capsule of the gland, but a few seemed to be inside it. They corresponded more in their appearances to chronic abscesses than to foci of fat necrosis. This fatty ensheathing tissue was especially abundant around the head and tail, but formed a thick covering all over, so that after its removal the pancreas itself was, if anything, smaller than normal. On section it presented a remarkable appearance, showing no look of pancreatic structure, except for about half an inch at the tail. In the head also there was an area about the size of a sixpence, or a little bigger, which looked like very fatty pancreas. All the rest showed a reddish-brown to black colour mingled with slatey-grey patches. The entire pancreatic tissue had here been destroyed by hæmorrhage, whose appearance suggested a multiple occurrence at different times. There were a few foci of softening, but it was mostly fairly firm. There were two thick-walled cyst-like cavities in the head of the organ filled with a putty-like substance. Fresh sections made after the specimen had been kept some time in spirit showed less difference in the suggested age of the hæmorrhages. The slate-coloured patches had disappeared, and the greater part presented the appearance of a comparatively recent hæmorrhage. There were several visible vessels filled with dark red clots. (Specimen shown.) The most noticeable points in regard to the other organs are that the liver and spleen both gave the iron reaction very distinctly, the heart showed a very distinct recent pericarditis, the kidneys a chronic nephritis, and the lungs several small foci of suppuration of an acute and subacute character. This indicated that a general sepsis had occurred, probably having its origin in the pancreas.

*Microscopic Appearance.*—Sections of the tail of the gland, its healthiest part, showed a good deal of chronic interstitial change and some degeneration in the pancreatic cells. Their nuclei stained fairly well, but the protoplasm was more granular and less distinct than normal. The interstitial change varied from slight to broad distinct fibrous bands (Microscope 1). Close to the margin of this tissue, where it bounded the hæmorrhagic part, several areas of small round-celled infiltration were seen among the gland cells (Microscope 2). Near them were some extensive and older interstitial fibrous bands rich in capillaries and blood sinuses (Microscope 3). Other parts at this bounding zone showed an abundant small round-celled infiltration practically obscuring the gland tissue, and close to it a patch in which the gland cells were completely necrosed, the cell walls and a little contained debris alone being left. Such patches were seen to be in close relation to duct branches (themselves considerably altered) (Microscope 4) and to vessels showing marked endarteritis obliterans

(Microscope 5), but no undoubted relationship could be established. Healthy gland tissue, or at least as nearly healthy as the healthiest part, was seen close to these areas, and no patch showed an early necrotic change in which the nuclei and protoplasm were still recognisable, but stained badly. Such areas were carefully looked for, and their absence showed that the process had been a very chronic one. The margin of the hæmorrhagic part showed fully necrosed gland lobules (outlines of cells and debris) separated from one another by a great deal of fat infiltration (Microscope 6), and the complete transformation of gland lobules into fatty tissue was also apparently taking place. A minor degree of this, where the individual cells were replaced by large fat cells, occurred here and there in the healthiest gland tissue present. Other sections were taken from the body of the gland at its margin, and showed purulent-looking centres lying mostly interstitially around necrosed gland lobules. This could be seen in different stages (Microscopes 7 and 8). Sections taken from the body of the gland, the hæmorrhagic part, showed a structureless material, composed of recognisable blood-clot in places, and of pigmented unrecognisable tissue in others (Microscope 9). The inflammatory interstitial tissue inside the gland and beneath the capsule contained numerous fine wavy and twisted bacilli, apparently all of one kind. No organism could be detected in the necrotic areas. The special changes to be noticed, then, are an inflammatory change, chiefly interstitial, abscess formations, necrosis, and hæmorrhage. It is possible that the inflammation first occurs, and leads to suppuration and then to necrosis and hæmorrhage, but I have not yet been able to establish this or any other relationship with certainty.

7. *Dr W. Russell* gave—(a.) A note of a case in which an ABDOMINAL ANEURISM had ruptured retro-peritoneally, and showed the specimen. The patient was a lady of 84 years. She was seized late at night with pain in the abdomen, and when her medical attendant, Dr Inkster, saw her soon afterwards, she was in a state of extreme collapse, from which she rallied under the hypodermic administration of ether. Dr Russell saw her with Dr Inkster the following afternoon. The patient had so far recovered that she was sitting up in bed, and there was no sign of collapse. She complained of discomfort in the left half of the abdomen. There was fulness anteriorly in the left lumbar region, extending up under the margins of the ribs; there was tenderness on pressure, so that deep palpation was impracticable. The fulness extended round to the lumbar region posteriorly, and here also there was tenderness on slight pressure. All the region referred to was dull on percussion, and continuous with the splenic dulness. The diagnosis was perforation within the abdomen, and from the seat of pain and the history of considerable stomach trouble, it was thought probable that the perforation was in the stomach at the

splenic aspect of the greater curvature. The prognosis was regarded as extremely grave, because of the probability of fresh extravasation occurring. The patient had a second attack of collapse, in which she died. Fortunately, permission to examine the abdomen was obtained, when it was found that the condition in the loin was due to an enormous mass of black blood-clot in front of and around the kidney, completely behind the peritoneum, but very markedly bulging forward, and carrying the peritoneum with it. The large mass of blood-clot was continuous by a narrow neck with the perforation in the aneurism shown, the aneurism being fusiform and of moderate size.

(b.) A note of a case of HÆMORRHAGIC PANCREATITIS which had been under his care in the Royal Infirmary. The patient was a man, aged 52, who died after a week's residence in hospital. The symptoms were very obscure. There had been a history of paroxysmal pain about the stomach and at the angle of the right scapula for five months. He had been off work for five weeks. He complained also of palpitation, pain about the heart, and weakness. For some days before admission he had nausea, vomiting, and delirium. He showed on admission purpuric spots on his lower limbs, which disappeared during the time he was in hospital. The temperature was subnormal, the pulse 84. There was some albumen in the urine, the heart was enlarged, and there was pericardiac friction. He was sometimes delirious and noisy, and was always rather "off his head." The red blood-corpuscles numbered 1,850,000 per c. mm., but showed none of the characters of pernicious anæmia. The most important lesion was found in the pancreas, the morbid anatomy of which Dr Leith had kindly undertaken to investigate. He submitted this brief note, as he thought it desirable to have reference made in the *Transactions* to a disease rarely met with. The full record of the case would appear in the *Hospital Reports*.

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## Meeting VII.—March 4, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

### I.—ELECTION OF MEMBERS.

The following gentlemen were elected members of the Society:—Harry Rainy, M.B. C.M., 25 George Square; William Alexander Mackintosh, M.B. C.M., 13 Abercromby Place, Stirling.

### II.—EXHIBITION OF PATIENTS.

1. *Dr Michael Dewar* showed a case of DIPHTHERIA treated by antitoxin, which, he said, he brought before the Society not because it was a case of diphtheria treated with antitoxin serum, but rather

because it had been diagnosed at first as one of croupous laryngitis. The child was *in articulo mortis* when the serum was introduced, and it might be said that the true nature of the disease was discovered by the treatment. He would not trouble them with all the details of the case at this time, but simply state the main facts as they occurred. On the evening of the 31st December last the patient was taken ill with a slight croupy cough, but was quite well otherwise, and running about the room. He was sent for to see him on the evening of the 1st January, *i.e.*, the next evening, and, after examination, made the diagnosis as he had stated, and put him to bed on biniodide treatment; applied poultices to neck and upper part of chest, and had the air of the room kept moist with the steamer. He was taking milk freely. He was in much the same condition during the next day, and had passed a fairly good night; but on the forenoon of the 3rd January the child was very much worse. He exchanged small doses of calomel for the biniodide every hour, and continued poultices and steam, and gave brandy and milk. He suggested a consultation. Dr Affleck was accordingly invited to see the case on the afternoon of the 3rd. However, he was not able to come till 9 o'clock the same evening, and at that time he concurred with Dr Dewar that it would be utterly hopeless to attempt tracheotomy or intubation, as the child was in a state of collapse. He was quite cyanosed, at times unconscious, almost pulseless, and without corneal reflex. After Dr Affleck had left the house, and while waiting for a little while to see the end, it occurred to him that the case might be one of diphtheria. Acting on the impulse, he went immediately for his syringe, and obtained a supply of British Institute serum from Duncan & Flockhart. At 10.30 P.M. he injected 10 c. c. underneath the skin of the abdomen. Next morning the child was still alive, and at 11 A.M. he injected another 8 c. c., with the result that from that time onward improvement set in and continued, till on the tenth day he was convalescent, with the exception, perhaps, of a little swelling of the submaxillary glands. He was now in good health, and a fair specimen of sturdy childhood.

2. *Dr James Carmichael* showed a boy, aged 9, suffering from CHRONIC PERITONITIS. The disease was steadily undergoing process of cure, as could be readily observed. He was admitted on Dec. 17, and had been nearly two and a half months in hospital. On admission his weight was 43 lbs., and now it was 49½ lbs. When admitted he had an extremely tubercular look, quite different from his present appearance. The abdomen was markedly distended, extremely tense, and difficult to palpate. They discovered the usual signs met with in such cases. There was a small amount of fluid in the peritoneal cavity. The exudate was chiefly of a solid nature, and he would just try to illustrate on the blackboard what they found. There were two large nodular masses and a number of smaller ones.

It was quite evident that the omentum and bowels were pretty well matted together with solid exudate. The question of surgical interference arose, but they kept him under observation for a short time, and he appeared to improve so rapidly that he (Dr Carmichael) did not think it necessary even to ask his friend Mr Bell to come and see the case. The boy was kept in bed for about six weeks, and had improved very much indeed, both in regard to general health and local condition. The abdomen was now not much distended, and was easily palpable, and there was no fluctuation. These cases of chronic peritonitis were of extreme interest, because of late years there was no disease in the treatment of which greater progress had been made, mainly on surgical lines. They now knew that many of them were greatly benefited by surgical treatment. Simply incising the abdomen and relieving tension, even although there were no great amount of fluid present, was quite enough to initiate a cure of the disease by stimulating absorption. He was old enough to have seen a great number of these cases both in hospital and private practice. He noticed the swing of the pendulum was going perhaps too far in the opposite direction. He had heard young practitioners say that they believed every case of this kind should be treated surgically. One object he had in showing this case was to prove what he had been fully convinced of himself, viz., that these cases, just like every other case, must be judged each on its own individual merits. Although he considered many of them were the better of surgical treatment, a large number did perfectly well without it. Extreme tension, whether from fluid or not, was an indication for surgical treatment; but in a case like this, where there was little fluid, and the tension was not great, there was no urgency for surgical interference. The temperature was slightly pyrexial at first, but for the last six or seven weeks had been normal. He did not think surgical treatment was in any way indicated, because such cases as this recovered, in his experience, probably within a couple of years, but required great care and attention to general and local treatment. As to local treatment he had something to say, because, taking a hint from the surgeons, he had lately treated four cases, two in hospital, and two outside, with constant massage of the abdomen. He did not know how the surgeons explained the effect of simple incision into the peritoneal cavity when there was no fluid to evacuate, or, at all events, very little. He supposed that in some way or other it stimulated absorption. No doubt incision was a most powerful and useful remedy, but there were other means of stimulating absorption without exposing the patient to the risk of a surgical operation, however slight. Another interesting point was the localisation of tubercular disease. How was tubercular disease localised in the abdomen so frequently? In this case all the other organs were quite healthy. Post-mortem examination in such cases frequently revealed extensive tuberculosis entirely localised to the abdomen.

3. *Mr Joseph Bell* said that by the kindness of the President he was at liberty to show a case which was not on the billet. It was one of considerable rarity, viz., that of a little boy, 5 years old, with CONGENITAL DISLOCATION OF BOTH HIPs. Most of them had seen cases; he himself had seen three or four. He wished to show this case, chiefly from the fact that the patient walked better than such patients usually did, and did not present the remarkable lordosis in which the sacral and lower lumbar vertebræ lay almost horizontal. He walked fairly well, and bent his knees fairly well as he walked. *Mr Cathcart* was kindly going to make a cast for the Museum of the College of Surgeons. These cases were accounted for in three ways:—(1.) It was held that they were damaged at birth; in certain breech cases damage was supposed to occur. (2.) That they arose from muscular spasm *in utero* drawing the bones out of joint. (3.) From imperfect development of acetabulum. He thought the last was the most likely. *Dr Leith*, however, would tell them. They would notice the way in which the pelvis was slung between the hips like a carriage on C-springs.

*Dr Leith* said *Dr Bell* had kindly referred to him. He might mention that he had lately shown to the Society a skeleton illustrating the condition. It was now in the Museum of the Royal College of Surgeons. The acetabula might be fairly perfect, but the hour-glass condition of the capsular ligament did not admit of an attempt at reduction. (*Vide* page 208, *Transactions of the Society*, vol. xiv. 1894-95.)

4. *Dr Adamson* showed, for *Dr Burn Murdoch*, a case of PATENT DUCTUS ARTERIOSUS. The patient was a boy, 8 years old, who had an attack of rheumatism three years ago. Since then he had not been so well, and was brought to hospital for treatment. There was considerable precordial pulsation, as the members of the Society could see; but he hardly suffered at all, and there was very little dyspnoea. There was no cyanosis or clubbing of fingers. The apex beat was most marked in the fifth interspace, one inch external to the nipple line. In the second interspace, immediately to the left of the sternum, a very pronounced thrill could readily be felt. A loud mitral systolic murmur, loudest in the mitral area, was heard, also a loud systolic and diastolic murmur in the aortic area, and up and down the sternum. *Dr George Balfour*, who had kindly examined the boy, had agreed in the diagnosis.

### III. EXHIBITION OF SPECIMENS.

1. *Mr Stiles* showed specimens of Prof. Tavel's ANTI-DIPHThERITIC SERUM, with Beck's SYRINGE for injecting it. He said he thought he might be pardoned for bringing this antitoxin before the Society. He did so partly because he knew the gentleman who

was responsible for it, and the institution where it was produced, and he hoped he would not be accused of trying to advertise his friend if he showed it. His friend's name was sufficient to exonerate him (Mr Stiles). He was Professor of Bacteriology in the University of Berne, and he had been authorized within the last few months to prepare a diphtheritic antitoxin for the State of Berne, and his investigations had been under the control of the State. A little while back he told him (Mr Stiles) that he had now got a considerable quantity of this material, had been using it for several months, and had found it very efficacious. It was only a day or two ago that Mr Stiles was talking to a British scientist on this matter, who said he did not think the British antitoxins in the market were always to be relied on. Mr Stiles was much struck with the way in which this antitoxin was preserved and sent out, and its admirable fitness for export. Most were sent out in corked phials, and therefore this was no guarantee that the serum would remain aseptic; but this serum was put into a cylindrical tube which was drawn out and sealed at both ends. Before it was sealed it was placed in an incubator for twenty-four hours. It was guaranteed aseptic. The tube was almost entirely filled with the serum, so that there was no oxygen to decompose it. It was very strong,—so strong that 1 c.c. contained from 100 to 200 antitoxin units, and 200 units, he believed, was the strongest anti-diphtheritic serum yet prepared. In the next place, the readiness and safety with which this could be made was remarkable. The mode of injection was as follows:—The drawn-out cylinder was first held vertical, then broken off and fitted on to a needle mounted on a bit of vulcanite, which again was soldered on to a bit of indiarubber tubing, and this was passed into the subcutaneous tissue. The serum was so potent that he believed  $\frac{1}{1000}$  or  $\frac{1}{2000}$  c.c. was sufficient to neutralise a lethal dose in an average-sized individual. Tavel recommended that it should be used as a preventive in cases exposed to infection. One hundred to 500 units injected would, he held, create immunity for from two to four weeks. For an adult he recommended 1000 units, *i.e.*, 10 c.c. He maintained that if the symptoms had not very materially subsided in the course of two or three days, another dose should be injected. Since receiving this he (Mr Stiles) had put the matter in the hands of a chemist who, he believed, had got the agency, and was told by him the other day that he had now got a good supply, *viz.*, Mr Stewart, No. 1 Lynedoch Place. It could be injected with an ordinary syringe. Prof. Tavel hoped soon to be able to supply an anti-tetanus.

2. *Mr Caird* showed a specimen of PERFORATION OF STOMACH, which, he said, illustrated a failure in surgery, but was very instructive. The patient was admitted to Mr Chiene's ward

suffering from symptoms of dyspepsia without any vomiting. The diagnosis was obscure. There was pain in epigastric region, but nothing was felt on palpation. He was in the ward for a few days. Then they concluded that probably he had carcinoma of pylorus, and one morning at 5 A.M. he awoke crying out with pain. Obviously perforation had occurred. He appeared moribund, with cold extremities, shrivelled fingers, sunken eyes; as usual in such cases mentally clear. Temperature was 95°, and pulse felt with great difficulty. Laparotomy seemed out of the question. They contented themselves with transfusing about 15 ozs. of saline solution into the veins of the arm, with the result that in two or three hours the temperature had risen to normal and the pulse was rather stronger, and accordingly they opened in the middle line. The peritoneal sac contained tea and various articles of food. On pushing the omentum to one side they found a large aperture towards the pylorus, through which fluid was gushing. The first thing done was to seize the tube of the irrigator, and insert that into the aperture, and thus the fluid was being syphoned out of the stomach, while at the same time they enlarged the incision and rinsed out the abdomen with sterilised fluid. By the time they got the abdomen tolerably free nothing was coming from the stomach. The question was, what was to be done with the perforation. It was impossible to suture. Every time they put in a stitch it cut its way out of the infiltrated tissue. They put an indiarubber tube right into the stomach, and then attempted gastrostomy after the method of Witzel, by which they hoped to shut off the whole aperture, making it continuous with the abdominal wall. But the difficulty here again consisted in the fact that the carcinoma lay at this point. One was tempted to resect the mass, which the condition of the patient hardly permitted. They did what they could to stitch the stomach, then seized the duodenum and stitched it across, and thus managed to make a pretty fair false œsophagus. There was no proper road through the pylorus. Mr Caird was not clear that he had stitched up the duodenum; he took whatever lay in the vicinity. They next did a gastro-enterostomy with Murphy's button. During the operation the house-surgeon transfused 10 or 15 ozs. of saline solution. The patient awoke perfectly comfortable; all his pain had departed. He went on fairly well, but his pulse was not as it should be. At night they began feeding him, and every time they fed him the pulse got stronger and better, and they left him at night feeling certain they had pulled him through. There was no sign of septic absorption; temperature was normal, and the pulse could be counted. In the evening he was again seized with symptoms of perforation. The tube had been passed through diseased tissue, which was devoid of elastic spring, and consequently had not grasped the tube tight enough. Food would therefore escape from stomach into the peritoneal sac.



It would have been better to have taken omentum and stitched it around the tube more accurately to the peritoneal surface. The event proved the truth of this, for he died on the following morning about nine or ten o'clock, and the sectio showed a fresh extravasation. In the specimen they could see the condition of the gastrostomy. They saw how very efficient Murphy's button had been. If greater care and time had been expended on the operation, and if perhaps before they fed him they had again washed out the stomach and got rid of irritating material, the result might have been very different from what unfortunately it turned out. The case served to suggest that in certain cases of perforation due to gastric ulcer, an excellent result might be gained by converting the lesion at once into a Witzel's gastrostomy and washing out the peritoneum and the stomach.

#### IV.—EXHIBITION OF PHOTOGRAPHS.

*Dr Gibson* said he wished to show two Photographs from cases of PARALYSIS OF OCULAR MUSCLES. They were both patients in the Deaconess Hospital—one present, the other past. The latter, a child of eleven months, was in for three days. It was difficult to get any connected history. The only thing was total paralysis of the third nerve of right eye. Death occurred two days after admission, and a post-mortem revealed hæmorrhage and neuritis of third nerve. The other case was at present under observation for tertiary syphilis. There was paresis of the whole of the muscles supplied by the third nerve of left eye. He thought at first that it was probably due to a gumma; but as there was rapid improvement under large doses of iodide, he now thought it was probably a meningitis affecting the nerve and passing away, although he admitted that a gumma often seemed, under the influence of this drug, to melt away like snow in sunshine.

#### V.—ORIGINAL COMMUNICATIONS.

##### 1. THE BLOOD IN DIABETES MELLITUS.

By ALEXANDER JAMES, M.D., F.R.C.P. Ed., Physician to the Royal Infirmary; Lecturer on Practice of Medicine, Edinburgh.

MOST observers who have investigated the composition of the blood in diabetes mellitus have been met by the interesting fact that, contrary to what pertains in most chronic ailments and debilitating conditions, in this disease the number of red corpuscles and the richness in hæmoglobin of the blood is not only not in any marked degree diminished, but is usually increased above the normal. *Leichtenstern* has referred to this, and gives the results of the observations of many others.

To account for this richness in corpuscles and hæmoglobin, he and the others have favourably regarded the view that it is

apparent rather than real,—that is to say, that it is due to the poverty of the blood in water, and its consequent concentration as the result of the polyuria.

Within the last three years I have had the opportunity of treating a greater than usual number of cases of diabetes mellitus, and I have taken advantage of this opportunity to look into this question as regards the blood.

Let me now direct attention to Table I., and first to the number of red corpuscles and percentage of hæmoglobin.

TABLE I.

No.	Red Corpuscles.	Hb. per cent.	Sp. gr. of Blood.
1	6,730,000	66	1056
2	6,100,000	61	1059
3	4,800,000	58	...
4	5,250,000	60	...
5	5,600,000	65	...
6	3,550,000	52	1054
7	...	...	1060
8	5,300,000	75	1056
9	6,280,000	118	1055
10	5,380,000	96	1055
11	5,564,000	112	1056
12	6,200,000	112	1057
13	4,460,000	55	1054
14	6,000,000	96	...

This Table shows that out of thirteen cases the corpuscles were over 6,000,000 in 5; 5,000,000 in 5; 4,000,000 in 2; 3,000,000 in 1. The hæmoglobin was over 100 per cent. in 3; 60 per cent. in 8; 50 per cent. in 2.

Bearing in mind that there are many sources of error in connexion with the estimation of corpuscles and hæmoglobin, I think yet we must conclude, as others have done, that in diabetes mellitus the tendency is rather to an increase in these constituents.

But, now, is this increase merely relative, is it the result of a lessening in the amount of water and consequent concentration? Were this so we should find a distinct increase in the specific gravity of the blood, but the last column of this Table, which represents the blood specific gravity (ascertained by Roy's method) shows no distinct increase.

I further endeavoured to get information on this point by investigating the effects on the blood of ingestion of food, and specially of water, and Table II. shows the results obtained in four cases:—

TABLE II.

FASTING.				AFTER FOOD AND DRINK.		
No.	Red Corpuscles.	Hb. per Cent.	Sp. gr. of Blood.	Red Corpuscles.	Hb. per Cent.	Sp. gr. of Blood.
1	6,232,000	110	1055	6,100,000	106	1055
2	6,316,000	118	1055	5,056,000	100	1055
3	5,564,000	112	1056	5,372,000	106	1055
4	6,208,000	118	1060	5,440,000	104	1054

In this Table are shown, first, the results of the examination of the blood between 5 and 6 A.M., the patients having had no food nor water for some nine hours previously; and, secondly, the results obtained at 10 A.M., the patients having all breakfasted and drank copiously of water two hours previously. One of them had also drank, about half an hour before his blood was tested, three-quarters of a syphon of potass water.

Bearing in mind that the estimation of the specific gravity of the blood, like that of the corpuscles and hæmoglobin, cannot be regarded as being absolutely correct, we yet must recognise that these results show that in diabetes mellitus there is not the evidence of a concentration or thickening of the blood which we should have suspected.

My contention, therefore, is that in diabetes mellitus the tendency to increase in corpuscles and hæmoglobin is independent of the proportion of the water in the blood, and I would assert that this increase represents a *vis medicatrix naturee*,—that is to say, an effort on the part of the organism to make up for the great loss of oxidizable material by an increase in the oxidizing power of the blood.

In support of this view I would adduce the effects of starvation on the blood and circulatory organs. In men deprived of food altogether it has been shown that the amount of corpuscles and hæmoglobin remains high for long periods, the weight of the individual diminishing rapidly. Further, an animal starved to death does not die until it has lost about  $\frac{4}{10}$  of its weight, and the proportionate loss in the different tissues and organs is as follows:—Fat, 97 per cent.; liver, 53 per cent.; muscles, 30.5 per cent.; skin, 20 per cent.; intestine, 18 per cent.; bones, 13 per cent.; nervous system, 3.2 per cent.; heart, 2.6 per cent.

The meaning of this is plain enough. A starved animal lives on, as it were, by feeding upon itself, and the tissues which can be dispensed with more easily are made use of to feed those which are of more value. The circulatory system is manifestly of the greatest value, for it is by it only that the transference can be effected, hence the little loss of weight which it shows in starvation.

But in diabetes mellitus we have another phenomenon which we can use in favour of our theory. This is the digestive power. All who are in the habit of seeing diabetic patients at their food cannot but conclude that, whatever else their ails may be, indigestion is not one of them. The amount of meat and green vegetables which they consume at meals is evidence of this, and is to be interpreted as indicating that Nature is trying to compensate for the loss of oxidizable material through defective secondary digesting or assimilating power, by increasing the primary digesting power. Further, we often meet with patients who in the pre-diabetic stage suffered frequently from dyspepsia, and who when diabetes had supervened no longer knew what indigestion was. I hold, therefore, that we have in this again an example of the *vis medicatrix nature*.

It would be interesting to know if in diabetes mellitus the state of the blood showed any relation to the course, favourable or unfavourable, of the disease. Except that it is in my experience the rule to find that the blood richness is most marked in the more vigorous cases, I can say nothing on this point. I have the idea, however, that when a diabetic is beginning fairly to lose ground, the richness of the blood, in corpuscles or hæmoglobin, will be found to be lessening.

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*The President* said he would be glad to hear any remarks on the condition of the blood in diabetes, and the theory which Dr James had evolved from that condition.

*Dr Gibson* said he thought this most ingenious paper of Dr James's must not be allowed to pass without an expression of admiration for the unusual combination of philosophy and physiology which Dr James always managed to introduce into any piece of work he brought before them. This paper recalled to his mind a short paper which had been published in the *Lancet* last year by himself, on the Increase of the Blood Corpuscles in Congenital Heart Disease. The fact was well known before, but in no work was the increase accounted for, or even attempted to be explained. He had undertaken to say it was a compensatory process. But Nature never compensated with direct intention. It was probably brought about thus: on account of the retardation in the blood current the blood corpuscles had less work to do, less oxygen to carry, less wear and tear; consequently they lived longer, and increased in number until equilibrium was brought about. This paper of Dr James's seemed to run on parallel lines; but the last observation had completely cut the ground from below his own feet if it were attempted to make the two things go on all fours. He would rather be inclined to think that the cases of greatest increase were just the cases in which the machinery was running down most rapidly. There was just one question he would like to put to Dr James,—What was the number of white blood corpuscles? Unless they had the relation of the

leucocytes to the hæmocytes, he did not think they were in a position to discuss the relationship of the red corpuscles to diabetes.

*Dr Noël Paton* said he had some diffidence in discussing this paper, because he had not made personal observations on the blood in diabetes. He had, however, made observations on the blood in other disorders. If his remarks were of the nature of destructive criticism he knew Dr James would not mind that, because they were good enough friends. In the first place, the clinical methods used in the enumeration of the corpuscles, estimation of the hæmoglobin and of specific gravity, were not quite satisfactory, especially if left in the hands of resident physicians; and for that reason one had always a certain doubt in accepting even enumerations of corpuscles. In regard to the number of red blood corpuscles, the differences were so manifest and so marked, that the idea of their being due to errors in method must be entirely dismissed. In cases of diabetes enormous variations are found. As Dr James pointed out, various authorities had described this. In some cases there was more concentration of blood; in other cases a smaller number of corpuscles. For estimation of hæmoglobin, he supposed Gowers's hæmoglobinometer was used. One knew how apt it was to go wrong in the wards of the Infirmary. Unless the same instrument was used and tested continually throughout the series, he thought they must doubt the results as to the hæmoglobin present. Then as to specific gravity, there again they had the same difficulty. He had not worked at this himself, but the late Dr John Balfour had used it a good deal in their laboratory, employing the same methods, and they found that unless they continually tested the solutions they were using they could not depend on the results. He did not know whether the solutions were tested in the ward with the pycnometer. The results of investigators showed that the specific gravity of the blood was in direct proportion to the number of red blood corpuscles. He thought the work of Sherrington and others had pretty clearly shown this. The amount of red corpuscles was so enormous as compared with the amount of proteids and amount of solids of serum, that it was the determining factor with regard to specific gravity. To what were they to ascribe this great increase in the number of red corpuscles? Dr James put forward the view of the *vis medicatrix nature* increasing them to keep up the oxidising power. In his (Dr Paton's) opinion the simple abstraction of water would account for this, always admitting that specific gravity results were to be accepted with some caution. As von Noorden pointed out, it was very possible that in diabetes they might have, as result of production of oxybutyric acid and so on, increased lymph production, and increased withdrawal of water from blood; but how were they going to get increased production of corpuscles in diabetes? In that disease

one of the sources of energy was not available, viz., the sugar, which was excreted. The organism must therefore depend for energy on proteids. Corpuscles were largely manufactured from proteids; and if they had an extra drain on proteids as a source of energy, how were they to get the amount of albumen required? He did not think that Dr James maintained that they had diminished destruction of corpuscles when they had increased oxidation. There was no evidence whatever that there was any increase of oxidation in the disease. On the contrary, they had evidence rather of diminished oxidation. If the proteids were used up, where were the proteids to come from for the hæmoglobin, which contained 96 per cent. of proteids? They had also to provide all the energy of the animal. He must say he could not see how Dr James was going to work in the theory of the *vis medicatrix nature* increasing the corpuscles, nor did he think he had demonstrated that it was due to anything more than either increased excretion of water or formation of lymph.

*Dr Leith* said he confessed his position was largely that of Dr Paton with regard to certain methods of examination of the blood, particularly the realm of the hæmoglobinometer. He had himself used this instrument a good deal, and had had opportunities of watching different observers use it. It was very difficult to get any two to agree upon any given colour. The same observer must carry through a series of observations as had been done by Dr James; but even then we were open to the fallacy that the appreciation of delicate shades of colour varied in the same individual at different times. Unless the greatest care was used, the hæmoglobinometer was an almost useless instrument, and even then but little reliance, in his opinion, could be placed upon its results. In diabetes there was a daily drain of water, and therefore an increased density of the blood. He would like to refer to Dr James's table giving the result of his observations brought about by an alteration in the diet of his patients. We had to remember that the daily drain of liquid had been going on in their case for a long time, and yet Dr James made only one observation upon the effect produced by the ingestion of a large amount of fluid. Did this single dilution not simply go to make up for a certain amount of the long-continued loss of fluid? Some of the figures, indeed, seemed equally explainable on this hypothesis. (Dr James: Certainly not. The water was running away as fast as we put it in.) He was glad to hear Dr James say this. It was an important point if he had found that the increased diuresis exactly compensated for the amount of fluid ingested. Dr James's use of the term *vis medicatrix nature* was one with which he (Dr Leith) was not familiar. Pathologists understood the term to mean an attempt on the part of Nature to get rid of the disease,

that living tissues generally are endowed with a power which may bring about a subsidence of abnormal changes, and a restoration to the previous normal condition.

*Dr Foulis* said he would just like to refer to one other point in the way of criticism. When *Dr James* described so graphically how one patient drank a whole large syphon of potash water, he supposed he overlooked the fact that each syphon contained about 20 grs. of bicarbonate of potash. That might account for the statement that the specific gravity was not in any way altered. Would the addition of 20 grs. not counterbalance the tendency to lowered specific gravity?

*Dr James* said that he did not think 20 grs. would have any apparent effect on the specific gravity.

*Dr Craig* said he suspected that there were not 20 grs. in a syphon. He had the highest authority—viz., *Duncan & Flockhart* and others—for saying that there were none of the potash waters sold with anything like the amount that the *Pharmacopœia* allowed.

*Dr James*, in reply, said that he had wished to elicit criticism, and he had got it. With regard to the condition of the blood in heart disease, *Bright's disease*, etc., observations had been made. *Leichtenstern* and others had referred to the condition in heart disease. There was straining through the tissues, and concentration. As regards the white corpuscles, he (*Dr James*) had measured them too, but did not put them down, as he was not sure of the result being very accurate. With reference to *Dr Paton's* remarks, he came to something more serious. They were all acquainted with the fact that the hæmocyto-meter and hæmoglobinometer and specific gravity apparatus did not give exact results, but they made these results as exact as they possibly could. There were, of course, chances of error; but the fact that the investigation had been made with the same apparatus and by the same man, who was thoroughly well up to his work, reduced that to a minimum. But the term *vis medicatrix naturæ* seemed to have acted as an irritant to their friends *Dr Paton* and *Dr Leith*. He had used this term advisedly, because some of their scientific explanations acted somewhat as an irritant upon him. There were many things that science could not explain. Why was it that in diabetics the digestive power was increased? In many diabetics, before the diabetes began, the digestive power was not so good, but afterwards it improved. A healthy man, or *Dr Paton* himself, he might venture to say, could not eat, digest, and enjoy anything like the quantity of food which a diabetic almost invariably can do. But let a healthy man fall into bad health and become a diabetic, his appetite will become greater, and his digestive power increased. Owing to the waste caused by the glycosuria, more food has to be taken into the body, and digested. Accordingly stomach, liver, pancreas, etc., are called upon to do more work, and they respond to the call. This is the *vis medicatrix*

*natura*. So, too, he held the blood was called upon for extra work, and it responded.

## 2. OPHTHALMIA NEONATORUM, ESPECIALLY IN REFERENCE TO ITS PREVENTION.

By W. G. SYM, M.D., F.R.C.S. Ed., Assistant Ophthalmic Surgeon, Royal Infirmary, Edinburgh.

THERE are two points in connexion with ophthalmia neonatorum to which I wish to direct your attention this evening.

The first of these—and on this portion of my subject I shall be very brief—is in regard to its treatment and *medical* prevention. There is a great similarity, but a great number of minor differences, between the various applications used in the treatment of the condition. Practically all surgeons and obstetricians seem to be agreed that what should be done is the free washing away of any discharge with an antiseptic solution, and the painting of the inflamed conjunctiva with a stronger antiseptic and astringent. By common consent, nitrate of silver solution is employed for the latter purpose in varying strengths of from 2 to 5 per cent.; but some employ the mitigated (Chevallereau, 1<sup>1</sup>), and a few even the pure (Desmares, 1) stick. As a lotion, corrosive sublimate is now the favourite; but others used by various surgeons are—chlorine water, which is highly spoken of, and probably with justice, by Burchhardt (2); biniodide of mercury, by Illingworth; quinine, 1.25 per cent., by Reich-Hollender (3); hydrastin (4), and more particularly formol. [The employment of formol for this purpose was, I believe, first advocated by Valude (5), who considers it superior to corrosive sublimate lotion.] Fromaget and Barabacheff (6) employ it in a strength of 1 to 2000 as a lotion, and in a solution of 1 to 200 as a pigment. In the adult the application of these stronger solutions is exceedingly painful. Kalt (1) of Paris warmly recommends Condy's fluid (1 to 5000), and appears to obtain good results, though others do not agree with him; but I suspect the explanation of his success is that by means of a neat little "laveur" he is enabled to attain to a more thorough cleansing of the conjunctival sac than is usually brought about. And this leads me to say that I believe the secret of successful treatment lies very much in this, that, provided your lotion be aseptic and non-irritating, it does not matter so very much what you use if you ensure that the pus is never left in contact with the cornea, and that none is allowed to remain in the conjunctival sac. For the purpose of attaining this end without dangerous pressure on the cornea various instruments are recommended, such as Doyen's (9) red rubber syringe, the Lagrange (8) and other hollow specula, and Kalt's laveur and head of water. There is, however, very little need of inventing instruments for the purpose, for an ordinary hairpin bent in a direction at right angles to its length

<sup>1</sup> The figures within parentheses refer to References at end of this paper.



answers every requirement, and is always to be obtained wherever there is a baby.

For my own part I employ frequent bathing—every two hours, or even every hour in a bad case—with lukewarm corrosive sublimate solution (1 to 6000), free smearing of the edges of the lids with iodoform ointment, and painting with nitrate of silver solution, gr. 10 or 15 to ℥j., every second day.

In regard to the medical prophylaxis, besides washing out the vagina with an antiseptic before delivery, it seems best to wipe the face of the child free of any maternal secretions before any water or antiseptic is applied at all, and while yet the lids are still anointed and protected by the vernix. After this is done, *but not till then*, the eyes should be washed with an antiseptic, and either a drop of nitrate of silver solution instilled according to Crédé's plan; or, as Tarnier (7) advocates now, the conjunctival sac should be dusted with iodoform powder. It is scarcely necessary nowadays to point out how very essential some such procedure is, nor to detain you with an account—though it is a very interesting subject—of the immense and beneficial change which has been brought about by Crédé and the universal adoption of his methods. The story of that highly gratifying chapter in medicine will be found in Fuchs's *Causes and Prevention of Blindness*. I need only say that Crédé and others reduced the proportion of cases suffering from ophthalmia neonatorum from about 10 per cent. to about .3, or even .1 per cent., by the use of antiseptic applications (8).

The second point with which I have to deal is the question of prophylaxis in the general. And first let me point out to those who have not studied the subject of ophthalmia neonatorum specially how very serious a matter this is. It has been estimated by different writers and investigators that of all cases of blindness which occur, about 30 per cent., more or less, are due to ophthalmia neonatorum. Some consider the proportion to be higher even than this. Thus Magnus (8) of Breslau says that 34 per cent., Katz (8) of Berlin 41 per cent., and Claisse (8) of Paris, that 46 per cent. of all cases of blindness are caused by this disease, which we must never forget is preventable or curable in 9 cases out of 10, perhaps in 49 cases out of 50.

Silex (9) states that there are 1800 cases of ophthalmia neonatorum in Berlin every year. Most of these, of course, are cured and leave no evil result, but to the blind population of Germany generally there are added 600 persons annually who have lost their sight from this cause.

There are at present in England and Scotland, according to the census returns for 1891, no fewer than 26,264 persons registered as blind; allowing—and it is a very generous allowance—that not 30 but 25 per cent. of these were due to curable or preventable ophthalmia neonatorum, then there would have been at this moment walking about and doing their own proper work, and

taking their own proper share in the joy of life and the prosperity of the nation, 6566 men and women, who are thus causelessly doomed to suffer under one of the greatest privations to which we are liable, and who are also more or less unproductive and a money-consuming burden on the community. Nor must the general practitioner turn round upon me and say that we specialists are to blame for this state of affairs, for the truth is that while the results of ophthalmia neonatorum bear so very large a proportion to the other causes of blindness, the cases of that disease brought to us are a mere drop in the bucket. I will not refer to the proportion affected of all children born in lying-in institutions, because, for obvious reasons, that would not be a fair criterion, but I give you the figures respecting all the births in and out of such institutions for a certain town. In the city to which I refer (Breslau), there took place within a certain period of time 12,000 births; 250 children, or 2 per cent., were affected with the disease (10). On the other hand, not more than 1·1 per cent. of all the cases which apply for relief at eye institutions are cases of ophthalmia neonatorum. By the kindness of the President and of Mr Berry I have looked over the records of 6000 out-patients at the Ophthalmic Department of the Royal Infirmary (3000 of the President's and 3000 of Mr Berry's), and found a percentage of only 0·37 of ophthalmia neonatorum. At my own Dispensary in Leith,<sup>1</sup> the social position of the patients attending which is, on an average, much lower, I find in 3000 cases a percentage of 0·7. As, however, I shall have occasion to point out to you presently, I think the proportion in the English institutions is probably a little higher. To this it must be added, that in speaking of the percentage among births I have spoken of *all* births; while in regard to cases reported to specialists I have given you the percentage among hospital patients only, for ophthalmia neonatorum is very rarely seen indeed among the better-to-do classes who consult one privately. From the disparity between the great frequency of this disease in the world and the relative rarity of its coming under the notice of specialists, you will gather, I think, two conclusions, viz., that *we* are not very much to blame for the patients who become blind, and that a very large percentage of the patients must get well either spontaneously or under very simple treatment.

I have referred to the fact that ophthalmia neonatorum is, in my opinion, commoner in England than it is in Scotland. This is to some extent borne out by the distinct difference in the relative proportion in the two countries of those who have been blind from birth,—for “blind from birth” in the census papers includes not merely the very rare cases of infants actually born without vision, but those much more frequent cases in which sight has been lost during infancy. In England there are 809 blind persons per

<sup>1</sup> Now the Eye Department of Leith Hospital.

million, and of these one in every six is blind from birth; in Scotland there are only 695 blind per million, and of these not more than one in 7·7 is blind from birth.

I cannot help thinking—and this is the chief consideration which led me to present this paper to you—that these facts must be taken in connexion with the very much larger proportion in England of births attended by midwives rather than by properly educated practitioners as compared with Scotland. In England, I understand, the proportion of births attended by midwives is rather more than one-half. I am not in a position to give numbers for Scotland, but I do not believe it is anything approaching to that (11). If this be so, then another evil yet has been traced to the action of ignorant midwives, and one which merits careful consideration. It is quite true that some midwives do recognise the importance of this condition, and advise their clients to consult a practitioner; but more than these are careless about the matter, and some are actively hostile to any such proceeding, and oppose it vigorously. It is looked upon by them as a reflection on their success in managing their cases, and they sometimes terrify their patients with a highly-coloured account of what will be the line of treatment employed towards the children. I have taken trouble to verify this myself, and you will find it remarked upon by other surgeons (8). In Schleswig, notwithstanding the fact that it is a punishable offence on the part of a midwife to fail to report a case of purulent conjunctivitis, the Committee of the Schleswig Association, who inquired into this matter, reported that the rule is not carried into operation at all satisfactorily (10).

Can we do anything, then, to bring about here a more satisfactory state of affairs? In certain States there are, as we have just seen, penal enactments directed against midwives who fail to report to proper authorities any case of purulent conjunctivitis in the newborn child. Austria and Switzerland took the lead in this matter in 1865; the midwife is obliged to call the parents' attention to the necessity of seeing a physician, and if they refuse to do so, she is compelled to report the case herself. The result of this has been that Horner was able to state that not a single case of blindness produced by ophthalmia neonatorum had been admitted to the Blind Asylum of Zürich for twenty years thereafter (8). The States of New York, Maine, and one or two others in America have adopted somewhat similar laws more recently. Thus Dr Howe got passed in the Legislature of New York State the following law:—"Should any midwife or nurse having charge of an infant in this State notice that one or both eyes of such infant are inflamed or reddened at any time within two weeks after its birth, it shall be the duty of such midwife or nurse having charge of such infant to report the fact in writing within six hours to the Health Officer, or some legally qualified practitioner of medicine of the city, town, or district in which the parents of the infant reside. Any failure to comply with

the provisions of this Act shall be punishable by a fine not to exceed 100 dollars, or imprisonment for six months, or both" (12).

This law, or one exactly resembling it, is now in force in eleven States of the American Union, representing a population of 28,000,000 persons. The States are—New York, Rhode Island, Maine, Minnesota, Ohio, Maryland, Connecticut, Missouri, New Jersey, Illinois, and Pennsylvania.

Personally I scarcely think that in this country such "grand-motherly" legislation is quite called for, but better education in this respect of those who are to have charge of women during puerperium should be aimed at, as well as the dissemination of knowledge throughout the general population. Several schemes for this purpose have been carried into operation in various countries. The Commission of the Schleswig Association, to which I have referred, agreed, among other matters, to petition the magistrates to issue to all parents registering the birth of a child a pamphlet entitled, "On the Danger of Inflammation of Infants' Eyes: Advice to Mothers who do not wish their children to become Blind."

In Havre a similar procedure is carried out. Fieuzeal takes time by the forelock, and wishes an "Avis aux Parents" to be given to each couple registering marriage. Colm wishes instruction in the question to be given in schools, the subject to come in as a branch of tuition under the heading of Anthropology. He admits, however, that it would not do to introduce the subject of gonorrhœa into the higher grade girls' schools!

In May 1885 a deputation of the Ophthalmological Society (13), headed by Mr Jonathan Hutchinson, who was then its President, waited upon a certain high official of the Local Government Board in order to endeavour to induce the Department to issue to all persons registering the birth of a child a printed slip having the following statement: "Instructions regarding new-born infants—If the child's eyelids become red and swollen, or begin to run with matter within a few days after birth, it is to be taken without a day's delay to a doctor. The disease is very dangerous, as if not treated at once may destroy the sight of both eyes." At first it had been also proposed that the registration officials should read these instructions to all persons in charge of women who were being attended when in labour by the medical officers under the poor-law. There are certain objections to this plan into which we need not enter, but there is less reason to oppose the handing of such a printed slip as was suggested to persons registering births, though it is quite true that very often the advice may come too late for *that* child, and may also occasionally be given to a totally irresponsible individual. Still, what is needed is diffusion of knowledge on the subject, and this plan works in the direction of attaining that end.

And even though Government may not see its way to adopt the suggestion made, more private and local distribution of such slips

as have been mentioned has been attempted here and there through the country with good effect. Thus I learn from a paper read before the North of England Obstetrical and Gynæcological Society by Mr Snell of Sheffield (14), that just such a card has been presented at the Sheffield Infirmary to every one bringing a case of ophthalmia neonatorum. And I have received information from Glasgow that a copy of a pamphlet, drawn up by Dr Russell, has for the last considerable number of years been handed to each couple registering the birth of a child. This pamphlet is printed at the expense of the Corporation, and is distributed without extra remuneration by the registrars at all but one of the district offices in Glasgow. I understand that Dundee thinks of following the good example, if indeed it has not already begun to do so.

In conclusion, in the event of the Midwives Bill becoming law, and being extended to Scotland, I would strongly urge that we should take great care that proper instruction be given to the women in regard to this vital point, and perhaps that a penal clause be added to prevent carelessness or improper behaviour in regard to it. And I would also suggest, whether that Bill (about which I express no opinion) becomes law or not, that, through our much respected Medical Officer of Health, the Corporation should be requested to issue a slip or pamphlet such as has been in use in other places. I think myself that the paragraph bearing on this point in Dr Russell's paper is very good, and might be adopted with advantage, but should be more in the form of Mr Snell's slip.

Dr Russell's paragraph runs as follows:—

*Eyes.*—The first thing to be done on the birth of a baby is to cleanse and gently wash the eyelids and thereabouts. If a baby's eyes run with matter and look red a few days after birth, take it *at once* to a doctor. Delay is dangerous, and one or both eyes may be destroyed if not treated immediately. The discharge is infectious.

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#### REFERENCES.

1. *Annales d'Oculistique*, December 1894.
2. *Archives d'Ophtalmologie*, March 1895.
3. *Ibid.*, November 1894.
4. Scott, *Ophthalmic Review*, December 1894.
5. *Annales d'Oculistique*, February 1895 and July 1893.
6. *Archives d'Ophtalmologie*, August 1895.
7. *Annales d'Oculistique*, January 1895.
8. *Causes et Prévention de la Cécité*, Fuchs.
9. Quoted in *Centralblatt für Augenheilkunde*, Supplement 1894.
10. *Centralblatt für Augenheilkunde*, April and May 1895.
11. Hart, Obstetrical Society of Edinburgh, *British Medical Journal*, ii. 1895, 133.

12. *Gaillard's Medical Journal*, New York, December 1894; see also *Journal Amer. Med. Assoc.*, December 1894 (Bettmann), and November 1895 (Howe).
  13. *Trans. of Ophthalmol. Soc.*, vol. v. 1885.
  14. *Luncct*, i. 1891.
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*The President* said he was sure all present must have listened with much interest to the paper Dr Sym had given them upon a disease capable of producing irretrievable mischief, ending in blindness of a large portion of the community. At the same time, he was inclined to think that the statistics alluded to in Dr Sym's paper referred to a time gone by to a certain extent. He did not think that recent statistics would bring out the same proportion. Much more attention had been directed to the subject in recent years. Judging from his own personal experience, he was inclined to say that cases were much less frequent now than formerly. One had only to think of the number of cases brought to the eye dispensary or the eye wards of the Infirmary thirty or thirty-five years ago to be struck with the fact that the proportion that came now was very much smaller, less than one to five of those brought formerly. He thought this condition was rectifying itself. The main point was to have attention drawn to the danger of the disease, so that suitable treatment might be adopted; and the chief difficulty, as Dr Sym had said, was that a large proportion of cases, chiefly in England, were attended by midwives, not practitioners; and midwives were not aware of the great danger. What was specially needed was the better instruction of midwives; there was no question of that. With careful treatment this disease should be eradicated, if suitable prophylactic measures were taken. With regard to treatment, which Dr Sym had glanced at, that was generally recognised as correct, and all medical practitioners were aware of it; but the great point was to see that the midwives were made fully aware of the necessity for professional advice. Government had been appealed to by the Ophthalmological Society of the United Kingdom with regard to this matter,—viz., such a notice as Dr Sym mentioned. It was hoped that such notice might be distributed to all concerned through the registrars of births, deaths, and marriages; but it was found that this would add so much to the duties of registrars, and the consequent expense would be so great, that the scheme fell through,—the Government could not undertake it.

*Mr Joseph Bell* said that papers which did not excite discussion were almost sure to be correct in every part. He was gratified by the extreme brevity and carefulness with which the statistics had been presented. The great thing the teacher could do was to be constantly repeating things; and they must constantly repeat to the public that midwives must be taught to get rid of this malady. It just required to be attended to.

*Dr Sym*, in reply, said he could tell them the exact percentage occurring at the present time among hospital patients. It was 37 per cent. for the last 6000 cases in the Royal Infirmary, Edinburgh. In Leith he found the proportion in 3000 cases to be exactly double that,—7 per cent. But any one who looked into a hospital in an English town such as Manchester would find twenty or thirty cases in a day of ophthalmia neonatorum.

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### Meeting VIII.—May 6, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. ELECTION OF MEMBERS.

Sir John Simon, K.C.B., D.C.L., LL.D., F.R.S., London, was elected an Honorary Member, and Prof. William Macewen, M.D., LL.D. University of Glasgow, was elected a Corresponding Member of the Society.

#### II. EXHIBITION OF PATIENTS.

1. *Mr Alexis Thomson* showed (*a.*) for *Prof. Annandale*—A man on whose face a PLASTIC OPERATION had been performed. The nose had been destroyed by a rodent ulcer of fourteen years' duration. The condition was shown in the photograph which was handed round. The cheek was not available, having been used previously. Mr Annandale accordingly liberated a central piece from the forehead, and slid it down, then separated the central piece of the upper lip from its posterior attachments and lifted it up to fill in the gap; when the central portion of the lip had soundly healed, the edges of the lateral portions of the lip were brought together in the middle line.

(*b.*) Two cases of PERFORATING ULCER OF THE FOOT. Curiously enough, he had had an opportunity of observing four cases within the last twelve months. The first case had had for three and a half years a perforating ulcer on the sole, one on the terminal phalanx of the great toe, another over the head of the third metatarsal. For this he stretched the posterior tibial nerve at the ankle, and at the same time scraped the ulcer. It was a case of tabes dorsalis, with very severe gastric crises and lightning pains in the extremities, locomotor ataxy not being fully developed yet. The second case had an ulcer on the plantar surface of the great toe, extending into the interphalangeal joint, and there was complete anaesthesia and analgesia in the area of the ulcer. There was absence of ordinary sensibility as high as the ankle on both sides, exaggerated knee-jerks, and ankle clonus. Both cases were instances of perforating ulcer depending on nerve lesions,—one spinal, the other peripheral.

(c.) A patient after operation for PERFORATED GASTRIC ULCER. The man had been troubled with his stomach since last April. He was walking down Pitt Street one forenoon, when he was suddenly seized with faintness and dizziness, and a pain which he said was somewhere about the umbilicus. He got home with difficulty, and was seen by Dr Sloan, who visited him again at night. He had been practically well before. He vomited a little that evening, and was sent to the Infirmary as a supposed case of obstruction; but on his being watched there until next morning without further vomiting, the diagnosis lay between perforated ulcer and acute appendicitis. There being nothing to confirm the idea of appendicitis, Mr Thomson rather inclined to perforated ulcer. On opening twenty-four hours after perforation, he found gas in the abdomen and much turbid fluid, curdled milk, and what appeared to be egg, and a considerable amount of yellow lymph,—over the adjacent stomach and liver a large accumulation, and beneath the great omentum and in the pelvis a still larger turbid accumulation. He found an ulcer on the anterior wall of the stomach near the lesser curvature and rather nearer the cardiac end than the pylorus, and each breath sending out gushes of fluid and curdled milk through the perforation. An œsophageal tube was passed and the contents of the stomach at once drawn off. The area of the ulcer was then invaginated. They washed him out thoroughly, pouring in large quantities of lotion, and splashing his bowels in fluid. A large glass tube was passed into the pouch of Douglas through a separate wound made below the navel. He made an excellent recovery. Curiously enough, there had been admitted to the wards another case of perforated ulcer last Friday, which he opened some six hours after perforation. The perforation in that case was in the anterior wall of the pylorus, and there was difficulty in closing it without obstructing the pylorus; but he also promised to do well.

2. *Dr James Carmichael* showed a little girl who, he said, had been under observation for about a year in hospital. It was a case in regard to which the diagnosis was difficult. She was seven years old. When admitted she could not walk, and was barely able to stand holding on to a chair. Before she had been long in hospital they noticed an improvement. About four months previously she had had an attack of bronchitis, and after being confined to bed for some little time, on getting up she was totally unable to walk. At present she walked with a peculiar waddling gait, and the peculiarity of the case was that the whole muscular system was feeble, and she had markedly flat feet. He had examined the case with the utmost care, and could find no evidence of disease of the central or peripheral nervous system or of the muscles,—in fact, they could not class it with any of the ordinary paralyses. The electrical reaction of the muscles was



normal. There was no sign or symptom which could be associated with disease of the nervous system,—nothing but extreme general muscular feebleness and want of tone. There was also very marked ligamentous laxity. She could bend her fingers back until they were at right angles with the hand, and the ligaments of all the joints were very lax. They had considered the possibility of pseudo-hypertrophic paralysis or progressive muscular atrophy, but the diagnosis of either of these diseases was excluded by the progress of the case. There was no atrophy of muscles; in fact, they were rather stronger now, and she had improved in strength in every way. It seemed to be a case of general muscular weakness and ligamentous laxity. There was no history of rickets in early years. Massage, gymnastics, and general tonic treatment had been carried out.

3. *Mr Wallace* showed two cases, both of which, he said, presented somewhat unusual features. The first was that of a man, 23 years of age, admitted to the Infirmary some months ago, suffering from tubercular disease of the tarsus, which necessitated amputation at the ankle-joint, strumous dactylitis of the little finger, the abscess requiring to be opened and the bone scraped. After being in hospital for some time he went out, and apparently became quite well. He came back one day, however, to let them see how he was getting on, when *Mr Wallace* noticed that his head was more asymmetrical than usual. He had a distinct swelling on the right side just over the anterior part of the temple. Asked if he had himself noticed it, he replied, not particularly, but that for a week or two he had had difficulty in keeping his hat on properly. He had no pain or discomfort whatever. *Mr Wallace* found fluctuation, and concluded that there was an abscess, probably tubercular in nature, and advised him to have it opened. He gave chloroform, made a free incision into the abscess, intending to remove the pyogenic membrane and treat it in the usual way. After opening, he introduced a finger, and found a sequestrum, which on being touched disappeared into a cavity. He now showed the sequestrum. It was of about the size of a florin, and consisted of the whole thickness of the skull. He put his finger into the opening through which it had passed, and found it entered an abscess cavity, which separated the dura mater for about  $2\frac{1}{2}$  inches antero-posteriorly, and nearly 2 inches vertically, while in depth between dura mater and skull it was rather more than 1 inch. It was a case of *tuberculosis perforans* (Volkman), and presented the usual features. The edges of the opening were perfectly smooth. It had given rise to no symptoms whatever. He thought the absence of cerebral symptoms was probably due to what they observed in cases of cerebral tumour, viz., that an opening having taken place into the skull, there was relief of pressure; second, the pressure was extremely gradual; third, it was exerted

on a part of the brain that gave little evidence of pressure, viz., the frontal lobe. The treatment was that usually adopted,—scraping the wall of abscess cavity, and very careful removal of caseous material and granular thickened tissue. The cavity was then stuffed with iodoform gauze. It healed up, and the man was now quite well, no bad symptoms having occurred.

The second case was that of a patient after trephining. J. H., æt. 36, a joiner, while carrying wood on his shoulder downstairs slipped and fell. He was found unconscious. He regained consciousness in a few minutes, and was carried to the Infirmary. On admission he was very drowsy, and could only be roused to answer questions with difficulty. There were several abrasions on the face, the left ear was partially torn off, and there was a small lacerated wound over the left parietal bone, which, however, did not go down to the bone. The pulse-rate was 42; the breathing was not stertorous. Pupils equal, and reacted to light. Mr Wallace saw the patient one hour later, shortly after he had been sick. The pulse was then 60; pupils equal; no paresis nor twitchings. The drowsiness was rather less marked, and the respirations were no deeper. There was no bleeding from either the ears or nose. It was decided not to operate. The head was shaved, and an antiseptic dressing applied. Next day the pulse was quicker, 80 per minute; respirations 28, temperature 102°. Some twitching of the right hand and right side of the face was present, and there was a question of paralysis of the left hand. If anything, he was more drowsy, but he answered questions. He swallowed fluids without difficulty, and had control over both the bladder and rectum. He was apparently in a condition of "cerebral irritation," so-called. On the following day, at 11 A.M., he was decidedly more unconscious, and could not be roused at all. The temperature had fallen to normal; pulse-rate, 76; respirations, 32. Twitching and paresis as before. At 1 P.M. he was more deeply comatose, and as he seemed to be hour by hour getting worse, Mr Wallace operated, and found the following conditions:—1. A large flap, with the wound previously referred to as its centre, was reflected from over the left parietal bone, when a fissured fracture was seen to run across the parietal bone into the coronal suture, which was loosened. A circle of bone was removed at the fissure, but there were no hæmorrhages. The dura was opened, but no clots were seen. 2. Another flap was now reflected from the right side, so as to expose the coronal suture, when it was found that the suture, from being merely loosened on the left side, was actually separated towards the pteron on the right. A circle of bone, including the suture and low down near the pteron, was removed, when a number of small extradural hæmorrhages were seen, but no large clot. The dura was incised, but although a director was passed in every direction, no clots were found. On neither side was the pia arachnoid anæmic; on the contrary, it was congested, and the

veins were dilated. For seventy-two hours after the operation the temperature was irregular, rising on the second day to  $101^{\circ}6$ ; the pulse-rate varied from 92 to 148, the respirations from 26 to 38. He remained unconscious until the evening of the fourth day from the accident,—*i.e.*, for fifty-six hours after the operation. On the third day after the operation the temperature became normal, and the pulse-rate and respirations gradually became slower. What was the condition in this case? No doubt at first there was concussion gradually recovered from. Probably, as the pulse became stronger, when reaction came on, small hæmorrhages were produced, which accounted for the increase of drowsiness on the day after the injury; but why did the drowsiness increase and unconsciousness result? Was it a case of passive œdema? The patient made an excellent recovery, and now, one month from the date of the injury, he is apparently perfectly well.

4. *Dr Norman Walker* showed a case of FACIAL WARTS, which the patient attributed to a "foul shave." He had communicated them from the chin to the scalp by scratching. There was a certain suggestion of molluscum, but he had removed one and found it to be an ordinary wart, consisting almost entirely of epithelium, with hardly any connective tissue in it.

### III. LANTERN DEMONSTRATION.

*Mr Caird* gave a Lantern Demonstration illustrating the PATHOLOGY OF THE APPENDIX VERMIFORMIS.

*The President* said he was sure the Society were all much indebted to *Mr Caird* for his interesting demonstration.

### IV. EXHIBITION OF SPECIMENS.

*Dr Norman Walker* showed two Microscopic Specimens of SKIN DISEASE. One was a section from the only case he had seen of ringworm of the scalp in an adult. The other was a specimen of the germs said to be responsible for eczema. They were distinguished by the fact that they were arranged differently from the ordinary pus organism, being always found in collections.

### V. ORIGINAL COMMUNICATION.

#### A FEW MORE WORDS ON STROPHANTHUS.

By *GEORGE W. BALFOUR, M.D., LL.D. Ed. & St Andrews, F.R.C.P. Ed.*

*"Fas est et ab hoste doceri."*

MR PRESIDENT AND GENTLEMEN,—I have to apologise for recurring to this subject so soon. I can only plead in justification the extreme importance of the matter, and the fact that the remarkable contrast that subsists between the action of strophanthus and that of digitalis has not, in my opinion, been sufficiently emphasized

upon former occasions. We have been asked to place our faith in strophanthus on the strength of a series of cases in which the employment of that drug was followed by the disappearance of dropsy, and in which the pulse-tracings showed an apparent improvement in the pulse-rate and in the blood-pressure. But, Sir, the like has happened before without either strophanthus or digitalis; nay, it happens daily even now. The symptoms that arise from heart-failure may be relieved in many ways, and recovery from them is no certain indication of the therapeutic value of any drug which may perchance have been coincidentally employed.

The myocardium is a hollow muscle which by its expansion and contraction assists and directs the circulation of the blood; in this action it is aided by valves so placed that, when closed, the whole force of the muscle is expended in propelling the blood onwards. Failure of any of these valves permits the blood to flow backwards as well as forwards, and so places the myocardium at a mechanical disadvantage in regard to the discharge of its function. But the heart works so well within its power that even when such an accident happens suddenly, there is not a falter in the action of the heart, nor any indication in the pulse-tracing of any interruption of the equability of the blood-pressure. This interference with the function of the myocardium ultimately leads to embarrassment of the circulation, but under favourable circumstances this does not happen for many a long day, so that even to old age such a valvular lesion may remain perfectly mute.

But whenever from overwork, privation, or debilitating illness, the general metabolism is interfered with, the heart suffers; its function is impaired. Like every weak muscle, the enfeebled myocardium reacts to stimuli irritably and irregularly, and does its own peculiar work imperfectly. The ventricles contract less effectually, the arteries are not so well filled, the blood-pressure falls, and venous remora is established, with all the evils that accompany this condition; soakage of all the tissues, with serous accumulation (dropsy) in one or more of the cavities of the body. If we put such a patient to bed, the mere adoption of the recumbent posture at once lowers the pulse-rate by an average of 12 (6-16) beats per minute, and in weakly, ill-fed patients the reduction is sometimes greater. This lowering of the pulse-rate amounts cumulatively to an increase of rest to the heart of over two hours in the twenty-four, and we can readily understand that this rest, coupled with warmth and better feeding, is sufficient to give such a fillip to the general metabolism as enables the heart to recover its tone and to discharge its function more perfectly. The blood-pressure rises, the balance of the circulation as between arteries and veins is restored, dropsy disappears, and the patient is re-established in comparative health.

The record of both the past and present history of medicine shows

that many, if not most, of our hospital patients require no other treatment, though recovery may be aided by the judicious use of purgatives, of diuretics, or of such drugs as either diminish the work of the heart or restore the tone of the myocardium. It is evident, however, that those drugs alone are of paramount value which increase the elasticity of the myocardium, and that this action can only be permanent when accompanied by a corresponding improvement in the general metabolism. Thus only can we arrest the organism in its downward career, and place it in such a state of metabolic equilibrium as will ensure the permanence of that improvement which is so often found temporarily to follow the employment of even imperfect and inefficient methods of treatment.

This important action is usually ascribed to all the members of the digitalis group; but though they all possess a certain similarity of action, they vary very much in activity, and they also vary considerably in regard to the structures upon which that activity is exerted, and these two factors have a most important influence upon their usefulness.

The action of *Digitalis purpurea*, from which the group derives its name, is in moderate doses to improve the elasticity of all muscular fibre, and as all the blood passes many times through the heart for once that it passes through any other muscle, the myocardium is powerfully affected, while the other muscles remain practically uninfluenced. In like manner the muscles of the arterioles are also early and powerfully affected, though not to the same extent as the heart, as only the blood going to the district supplied by these vessels passes through them.<sup>1</sup> The increased elasticity imparted to the myocardium by digitalis enables it to expand and to contract more perfectly, and as, owing to the increased elasticity of the muscles of the arterioles, the arteries empty themselves more slowly, the blood accumulates within them, the blood-pressure gradually rises, and in accordance with Marey's law<sup>2</sup> the heart's action is slowed. No doubt this is partly due to the action of digitalis on the vagus nerve, but the action on the muscles is sufficient to account for all the phenomena observed, and it is with it we are chiefly concerned. The result of this rise in the blood-pressure is that the secretions are improved, and all the tissues of the body, amongst them the myocardium, are fed with richer blood at a higher pressure, so that metabolism is more perfect and every function more efficiently performed. As the blood accumulates within the arteries the veins are correspondingly emptied; the serous soakage of the tissues is reabsorbed, and the excess of water

<sup>1</sup> Stockman, *New Official Remedies*, p. 58. London, 1891.

<sup>2</sup> *Physiologie Médicale de la Circulation du Sang*, p. 206. Par le Dr E. J. Marey. Paris, 1863. Le cœur bat d'autant plus fréquemment qu'il éprouve moins de peine à se vider.

that thus gets into the blood is removed by the kidneys, so that for a time the urine is increased.

The improved metabolism of the myocardium enables it to discharge its function more perfectly, and to resist successfully all those deteriorating influences to which a weak heart succumbs, any hindrance to the circulation is fully compensated, and despite the existence of a valvular lesion the individual may descend into the vale of years wholly unconscious of the possession of a heart.

All the benefits we obtain from the use of digitalis are inseparably connected with its tonic action; they flow from the power that digitalis has of increasing muscular elasticity, and the improved metabolism of all the tissues, but specially of the myocardium, that follows this. Digitalis is no opium to the heart; it does not relieve by narcotising, but it soothes cardiac irritability by strengthening the cardiac muscle, and it assuages cardiac pain by improving cardiac metabolism, failure of which has been the cause of the pain. These benefits are readily obtained by very moderate doses of the drug, and though great benefit may at times be more rapidly attained by the judicious administration of larger doses, yet the long continuance of even small doses is often followed by the very best results; while the abuse of the drug, so frequently accompanied by distressing, if not alarming symptoms, proceeds upon an entire misconception of the true action of digitalis.

There is only one other member of the digitalis group that has succeeded in obtaining special recognition from cardiac therapeutists, and this is the *Strophanthus Hispidus*, but the action of this drug is so entirely different from that of digitalis that it is difficult to see how there can be any comparison between the two. In Africa, its native habitat, strophanthus is employed as an arrow poison, and the natives have so great a dread of it that when told that the seeds were employed as a medicine in this country, "they expressed the opinion that the English must be mad to employ so poisonous a substance for medicinal purposes."<sup>1</sup> The action of strophanthus on the heart is twofold: in small doses it arrests the heart in diastole, and in large doses it arrests it in systole.<sup>2</sup> The diastolic type of action seems to be that which alone is useful therapeutically; in it the diastole is prolonged and the contractile energy of the myocardium at the same time increased. The type is thus that of bradycardia, a slow-beating heart sending forward a large blood-wave with each contraction,—not a type of heart usually associated with much vigour of the circulation. This type of heart action is produced only by small doses; and the excessive energy of the drug, the many preparations of strophanthus in the market, of varying composition and

<sup>1</sup> Fraser on "*Strophanthus Hispidus*," *Transactions of the Royal Society of Edinburgh*, vol. xxxv., part 4, p. 974.

<sup>2</sup> *Ibid.*, vol. xxxvi., part 2, p. 401.

strength, must introduce an element of uncertainty in its employment that cannot be devoid of anxiety. Strophanthus acts three thousand times more powerfully on the heart than digitalis;<sup>1</sup> so that if some still dread digitalis as a dangerous cardiac poison, what must be their feelings towards strophanthus. And if it be difficult to select an appropriate dose of a drug of but moderate energy and only one action, how great must be the difficulty in the case of a drug of such excessive potency and possessing a twofold action on the heart. Indeed, whether we regard the uncertainty of its direction or its excessive energy as a poison, it seems difficult to formulate a more forcible indictment against any drug than that presented by a bare statement of the facts recorded against strophanthus by its most able investigator and most ardent advocate.

But this is not all. Digitalis, as we have seen, acts equally upon all muscular fibre, and only more powerfully upon the heart and muscles of the arterioles because these organs receive from the blood within a given time a larger dose of the drug than the other muscles. The conditions as regards the circulation are permanently similar; and if strophanthus acted on all muscular fibre after the manner of digitalis, there would be a similar ratio between the action of the two drugs on the heart and on the arterioles, but this is not the case. Strophanthus acts three thousand times more powerfully on the heart than digitalis, but it acts one hundred times less powerfully than digitalis upon the muscles of the arterioles.<sup>2</sup> There is thus no similarity whatever between the action of the two drugs. From the entire absence of any appreciable action by strophanthus on the arterioles, the blood flows freely from the arteries into the veins, and any little rise of blood-pressure there may be is entirely due to the systole of the heart. The typical (therapeutically induced) strophanthus heart has a prolonged diastole, so that the arteries have a longer time than usual to empty themselves, and as the ventricle has been filling during this period, its systole sends an unusually large blood-wave into unusually empty arteries—precisely what happens in a case of bradycardia. The momentary rise of the blood-pressure passes rapidly with the blood-wave down the unfilled arteries; it does not, therefore, persist so long as the ventricular systole, and is consequently of shorter duration than even the third of a cardiac cycle. So evanescent a rise in blood-pressure can, however, have no appreciable effect either on general metabolism or upon that of the myocardium. Herein the action of strophanthus differs most essentially from that of digitalis; and from the absence of any improvement in the metabolism of the myocardium, the stimulating action of strophanthus must tend still further to exhaust it instead of strengthening it.

<sup>1</sup> Fraser, *op. cit.*, vol. xxxvi., part 2, p. 403.

<sup>2</sup> *Ibid.*, pp. 438, 453.

Remembering this, the remarkable twofold action of strophanthus on the heart is easily understood. In large doses it exerts its own poisonous energy unchecked and forces the heart into fatal systole. In minimum lethal or so-called therapeutic doses strophanthus exerts a less powerful stimulation on the myocardium, with a type of action in which slowing of the rate is produced by prolongation of the diastole, and by occasional pauses in extreme diastole, both auricular and ventricular expansions and contractions being at the same time increased.

This tendency to arrest in diastole is not due to any action on the cardio-inhibitory apparatus, because paralysis of the intracardiac terminations of the vagus by atropine does not prevent its occurrence. It cannot be due to the action of the drug on the myocardium, as that would imply that in small doses strophanthus is capable of exerting an action on muscle diametrically opposite to that which it undoubtedly exerts in large doses. Accordingly Fraser has suggested that this diastolic type of action is due to a weakening of the excito-motor (katabolic) nerve structures. But so far as I am aware there is no evidence extant to show that even section of the sympathetic, excito-motor, or katabolic nerve does slow the heart's action, or in any way promotes slowing of the heart or its arrest in diastole. Before accepting "weakening of the excito-motor nerve structures" as explanatory of this peculiar type of action, it must therefore first be shown that this "weakening" does occur, and, second, that it is capable of acting as Fraser has suggested. There is, however, another explanation of this diastolic type of action which is in complete accordance with the facts, though it is not an explanation that can be regarded as very comforting to those who regard strophanthus as an important therapeutic agent. Strophanthus undoubtedly exercises a powerful stimulating action on the myocardium, an action which is three thousand times more powerful than that of digitalis, which forces the heart into energetic movements of expansion and contraction, but which is not associated with any important rise of blood-pressure, and is unaccompanied by any improvement in the metabolism of the myocardium. A large dose of the drug speedily forces the heart into fatal systole, but a minimum lethal or so-called therapeutic dose causes a slow action of the heart with prolongation of the diastole. During this prolonged diastole two or three auricular contractions overdistend the ventricle, which then empties itself with all the force it can still command. The contractility of the heart is not destroyed, but it is exhausted, and requires a more than usually powerful stimulant to rouse it to action. Rhythmic contractions occur spontaneously at long intervals under the influence of overdistention, even during prolonged standstill in diastole the ventricle can still be made to contract perfectly by mechanical irritation, and contractility only finally disappears when, after a long period of suspended action, the overdistended



ventricle slowly and finally empties itself, shrinking to normal or smaller than normal dimensions.<sup>1</sup> Such a heart affords a typical example of a heart dying from exhaustion, the cause of which is not far to seek. Forced into increased energy in its movements, without any corresponding improvement in its metabolism, the heart has been compelled to draw upon its reserve, and when this fails death from exhaustion speedily follows. When death occurs in fatal systole after the administration of strophanthus, the cardiac muscle is found to be acid and to be so profoundly modified that it passes at once into a state indistinguishable from that of *rigor mortis*,—it is fatally poisoned. On the other hand, when a minimum lethal dose is administered the contractility of the heart is not abolished, and the myocardium still remains neutral or alkaline to the last.<sup>2</sup>

It has been claimed that strophanthus is not cumulative. As we have just seen, it may be injurious or even fatal without warning, and from this point of view there may be worse things than the warning we get of approaching saturation from those symptoms we are accustomed to ascribe to accumulation.

It has also been claimed that the absence of any action on the arterioles is to be reckoned to the advantage of strophanthus in those numerous cases in which vascular stimulants require to be conjoined with digitalis to prevent any untoward rise of blood-pressure. But apart from those evils already referred to, which are caused by this absence of action on the arterioles, we know that though improvement in the cardiac metabolism is the one thing needful in all cases of heart-failure, it is of paramount importance in the class of cases just alluded to, because in them the heart-failure is due to the inability of the heart to cope unaided with the obstacles opposed to the exercise of its function. The employment in such a case of a drug like strophanthus, whose action is to stimulate and exhaust the reserve energy of the heart, must tend still further to enfeeble that organ, and persistence in its use must be most damaging to the integrity of the organism. On the other hand, the judicious combination of vascular stimulants with so powerful a cardiac stimulant as digitalis enables us so to regulate the blood-pressure as to keep it equable and efficient throughout the whole cardiac cycle, to the manifest advantage of the cardiac metabolism, and this, too, without any risk of the blood-pressure ever becoming so high as to be injurious.

The action of strophanthus is thus, like that of all its congeners of the Apocynaceæ, that of a cardiac poison, and not a cardiac tonic. In large doses—though from the energy of the drug such doses may be really small—it forces the heart into a fatal systole. In smaller doses it stimulates the heart to increased action, and

<sup>1</sup> Fraser, *op. cit.*, vol. xxxvi., part 2, p. 419.

<sup>2</sup> *Ibid.*

by calling on its reserve of energy without improving its metabolism it causes death in diastole from exhaustion, and the more feeble the heart is the greater the risk attending this peculiar action. Strophanthus may occasionally be of use in cases of ruptured compensation, but any assistance which it gives is at the expense of the cardiac reserve, and the patient is only saved from serious disaster by the benefit he has derived from rest, warmth, and nutritious food,—that is, by the improvement in his environment generally.

Strophanthus is thus at all times an uncertain and dangerous drug to employ, and one entirely unworthy of being called a remedy.

It is quite otherwise with digitalis. This drug does not act by calling on the reserve of cardiac energy; but by improving the metabolism of the organism generally, and especially of the myocardium, it adds to this reserve, and aids any improvement in the environment, not only to tide over a temporary disability, but also to restore the myocardium to such a condition of comparative health as will enable it to withstand all the deteriorating influences to which it may be exposed.

It is difficult to conceive a greater contrast than that which subsists between the action of these two drugs, and the more carefully the actions of both are considered, the more markedly these differences will be found to be accentuated.

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*The President* said he would be glad to hear any remarks from Members present on this most important communication on the action of digitalis and strophanthus on the heart.

*Dr Graham Brown* said he rose simply to say he was quite sure the Society wished to thank Dr George Balfour for the very able and excellently clear statements that had been made. He was quite sure they would recommend themselves to all who had attempted clinically to make use of the drug.

*Dr James Ritchie* said he rose to thank Dr Balfour for having formulated the reasons for digitalis being so very much more useful than strophanthus. He would have liked at the same time had Dr Balfour given his experience of strophanthus, because it would have been satisfactory to have heard a little more about the cases in which that drug had been used with benefit. He had often remarked to Dr Balfour that in cases in which he wished to obtain a rapid action he could get it by strophanthus much more readily than by digitalis. He found, however, that in continuing with strophanthus he did not get beyond a certain point of improvement, whereas by long-continued use of digitalis he got a very marked continuous benefit, so that after months the heart was left in an improved condition. The patient was benefited only for the time being by strophanthus. In one class

of cases he had not found digitalis of use, even a very distinct disadvantage, because of its action on the bloodvessels, viz., those accompanied by angina, and in those cases he had found strophanthus beneficial; but then one had to acknowledge that it was necessary to go on continuously with the drug. There were certain cases, however, in which they had to go on using digitalis continuously. He should like to have Dr Balfour's opinion of the relative merits of the drugs in aortic incompetence. From what Dr Balfour had stated to-night he felt encouraged to go on using digitalis.

*Dr Leith* said he thought the Society should not allow this discussion to languish. He hardly cared himself to speak except to express the hope that some other Members would do so. He did not hear the beginning of Dr Balfour's statement, having been unfortunately kept by a case as to which he had great doubt whether he should give strophanthus or digitalis. He gave strophanthus—he hoped he was not wrong. From what he had heard he did not think Dr Balfour had proved his contention at all. That he had proved some of it they might allow. Probably digitalis was the better drug of the two, but that it was always superior to strophanthus he thought might be doubted. They all listened to Dr Balfour's opinions with pleasure and respect, but even from him they could not accept statements, however well expressed, as convincing unless supported by sufficient proofs, and this he thought Dr Balfour had failed to give. There were many points touched upon by Dr Balfour to which he would like to draw attention, but he would content himself by referring to his statement that single doses of strophanthus did not stimulate the heart to contraction, but that the result of many continued doses was a single huge contraction, which left the heart exhausted. Could that possibly be true? His own experience, gained at the bedside and in the post-mortem room, supported another and more beneficial action. He had seen cases in which the heart was found post-mortem to be in a marked state of fibroid or fatty degeneration, which during life answered admirably to every dose of strophanthus. He could particularly recall one case which was enormously benefited by it. The patient had a very weak heart, and was frequently threatened with complete cardiac failure. He improved greatly under strophanthus, as shown alike by his subjective sensations, the pulse tracings, and the physical examination of his heart. It enabled him to tide over difficulty after difficulty, and apparently kept him alive much longer than had been hoped. On his ultimately succumbing, it was found at the post-mortem that the cardiac muscle showed a high degree of fibroid degeneration. Could Dr Balfour's contention be right in the light of even one such case as this? If the administration of strophanthus had merely enabled the heart to contract at intervals once more powerfully than usual, and then left it exhausted, would it not have

injured instead of greatly assisting him? Could he have gone on for six weeks or two months taking a cardiac poison when his heart was, unaided, unable to do its proper work? He feared he must object pretty strongly to this part at any rate of Dr Balfour's dictum.

*Dr Lockhart Gillespie* said he did not want to enter into the tilt against strophanthus, but would like to ask one question. He did not think Dr Balfour mentioned, or only shortly, the diuretic action of strophanthus. He thought Prof. Fraser had shown that it had a strong diuretic action. The fact that it did not raise arterial pressure, while, on the contrary, the diuretic action of digitalis was largely due to increased pressure in the blood-vessels of the kidneys, might be of great use in treating many cases. He would ask Dr Balfour if he had found many cases which could not tolerate very small doses of strophanthus, and yet could tolerate digitalis, and this apart from the action of these drugs on the heart. He had lately seen a case of weak heart with a practitioner in town. They gave small doses of strophanthus. After receiving two doses the patient became very sick. He changed it to the tincture of digitalis, which was tolerated. One got good out of strophanthus by beginning with it and carrying on with digitalis afterwards. He did not think Dr Balfour mentioned the cumulative action of digitalis. What was a small dose for one patient was not small for another; and if they were not careful with digitalis, untoward symptoms in aortic incompetence or other heart disease might occur.

*Dr Balfour*, in reply, said that he looked upon strophanthus as so dangerous a poison that he was not disposed to recommend its use under any circumstances. In regard to its supposed diuretic action, he begged to remind the Fellows of the Society that in heart affections diuresis occurred, under favourable circumstances, without the administration of any drug whatever. In the course of a few months he hoped to bring before them a whole series of cases in which no drug whatever had been administered beyond a free supply of potash water. Yet in them the pulse-tracings would be found to show a gradual rise of blood-pressure, while copious diuresis would be found to accompany the general improvement of the condition. It was a mistake to suppose that because a certain drug had been given, all the benefits that followed were due to it alone. Strophanthus did not improve the cardiac metabolism, and acted only by calling on the cardiac reserve. It was therefore always a dangerous drug, and ought, he thought, to be expunged from the Pharmacopœia. In regard to Dr Ritchie's notion that digitalis was not always useful in angina, angina was only a symptom, and arose from such various causes that digitalis could not be expected to be useful in all. When, however, it arose from cardiac debility no drug was so useful as digitalis. It was *facile princeps*. Of course, when angina occurred with a feeble

myocardium and a high blood-pressure, digitalis had to be conjoined with a vascular stimulant; if this were not done harm instead of good would result. With this adjunct the metabolism of the heart was improved, the debility of the myocardium remedied, and the angina cured, as he had seen in scores of cases. As for the cumulative action of digitalis, every drug was cumulative. Even Epsom salts might be administered in cumulative doses till they brought on diarrhœa. Digitalis was also cumulative, and its symptoms varied with the dose. One old friend of his, an old gentleman over 70, had a difficulty in going up even a short stair from a slightly dilated heart. This patient took a single grain of digitalis every night for over two years. At the end of that time the only sign of accumulation that appeared was a bigeminal pulse, and this peculiar symptom rarely troubled him except after food, and chiefly after breakfast. The digitalis was stopped, but it had done its work; and the old gentleman could now run up three or four pairs of stairs without the slightest difficulty. He recommended all those who had any leanings towards strophanthus to study Fraser's papers in the *Transactions* of the Royal Society of Edinburgh, and he thought if they did so their ideas in regard to strophanthus would undergo considerable modification, and digitalis, which had been gradually winning its way to the front during the last 150 years, and was only now beginning to be recognised as *facile princeps* of all cardiac tonics, would have some chance of having its actions more carefully studied. It was doubtful if there was another drug in the Pharmacopœia which was so useful. Certainly within its own range there was none more so, nor any whose action could be more easily regulated and controlled.

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### Meeting IX.—May 20, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. EXHIBITION OF PATIENT.

*Mr Joseph Bell* showed a case of GUNSHOT WOUND. His friend, the subject of this accident, had kindly come to show his arm; and here also were some Röntgen photos. He was shooting pigeons two months ago, when his rifle accidentally went off, and he was shot in the forearm just above the wrist. There was considerable hæmorrhage from the external aperture, and considerable shattering of bone. Mr Bell cut down and removed a loose piece of the radius, put the limb into position as well as possible with a good deal of trouble, and got it sweet. Fortunately

it remained sweet. There was hæmorrhage from the posterior interosseous, but he plugged with iodoform gauze. It went on absolutely uneventfully without discharge, and healed quite rapidly and thoroughly. He thought the Society would be interested in the fact that the patient had got fair movement of his arm. Rotation was not quite complete, but the movement of the fingers was perfect. It was only about a couple of months since it happened, and it was improving in every way. Dr Dawson Turner had taken the photos. They had not accounted for the whole of the bullet. Some of it came away at the time of the injury,—the fragments of which were shown. The small pieces shown by the photos. had remained in the limb. A considerable amount of callus had been thrown out, and the end of the radius was broadened, notwithstanding the loss of substance that took place after the accident. A large piece of bone removed was shown. The patient had now a good grip with the affected limb. About a third of the bullet was taken out, and the rest seemed to have passed out at the time of the injury.

## II. DEMONSTRATIONS.

1. *Dr Norman Walker* demonstrated a handy method of using the FLASH LIGHT IN PHOTOGRAPHY. He said that what he wished to show was a powder adapted for taking photographs instantaneously. The value of photography had been demonstrated, and this method was exceedingly simple, and enabled one to take a photo. at any time of the year irrespective of the weather. Like other members of the Society, he invested at one time in an expensive apparatus which cost a guinea. This flash light, devised by Dr Brock of Manchester and Dr — of New York, was cheap and very efficacious. The materials were gun-cotton and this mixture of chloride of magnesium and chlorate of potash. (Dr Walker now applied a match to some of the powder, which had been placed on an ordinary fire-shovel, and thus demonstrated the flash.) The momentary flash was sufficient to take the photo. He had brought one of the photos. to show. It did not matter although the person who was being taken jumped. The light was held exactly over the camera.

2. *Dr A. B. Gils* showed a very simple and very cheap form of FLASH LIGHT APPARATUS. The whole cost less than sixpence. It consisted of a longish clay pipe with a small bowl, and having an indiarubber tube and ball attached to the mouthpiece. The magnesium powder was placed in the bowl of the pipe, and could thus by compression of the indiarubber ball be blown into the flame of a spirit lamp set in front of the apparatus.

## III. ORIGINAL COMMUNICATION.

## ON THE TREATMENT OF INOPERABLE CASES OF CARCINOMA OF THE MAMMA: SUGGESTIONS FOR A NEW METHOD OF TREATMENT. WITH ILLUSTRATIVE CASES.

By GEORGE T. BEATSON, B.A. (Cantab.), M.D. (Edinburgh), Surgeon to the Glasgow Cancer Hospital; Assistant Surgeon, Glasgow Western Infirmary; and Examiner in Surgery to the University of Edinburgh.

MR PRESIDENT AND GENTLEMEN,—I have no doubt it has fallen to the lot of nearly every one here present to have been consulted from time to time by patients suffering from carcinoma so widely spread or so situated that it has been quite apparent that nothing in the way of operative measures could be recommended. Such cases naturally excite our sympathy, but they also bring home to us the fact that once a case of cancer has passed beyond the reach of the surgeon's knife, our curative measures are practically *nil*, and "that, whether the case advance with giant strides or with slow and measured steps, the result is equally sure and fatal." Of late, owing to my taking up the work of surgeon to the Glasgow Cancer Hospital, I have seen a considerable number of such cases, and an opportunity has been furnished me of working out a line of treatment which I am not aware has been as yet tried by others, and which is founded on a view of the etiology and nature of cancer which is entirely opposed to the local parasitic theory of the disease, and which seems to me to offer a more reasonable explanation of it. As these inoperable cases of cancer may be arranged into two groups—first, those which have been operated on, but in which, sooner or later, there has been a recurrence, or, as it should perhaps be better expressed, a reappearance of the disease; and, secondly, those in which no operation has been attempted, but in which, when they first present themselves, the disease has progressed so far that no local removal could be attempted—I shall bring forward three cases, one of which is illustrative of the first group, and the other two of the second.

The first case, then, that I wish to bring under your notice is that of Elizabeth B., who consulted me on 11th May 1895, at the Glasgow Cancer Hospital, bringing me the following letter:—

" 37 APSLEY PLACE, 6th May 1895.

" DEAR DR BEATSON,—The bearer, Mrs B., is, and has been suffering, I fear, from a malignant breast. She has been in the Royal Infirmary before she came to me. My own opinion is that nothing can be done for her; but as she is a woman of great courage, you might have a look at it for my sake, and perhaps you can order her something in the way of dressing. Even this little will be accepted by her as a great deal.—With kindest regards, yours very truly,

" JAMES W. WALLACE."

The history she gave me was that she was 33 years of age, married, and the mother of two children, the oldest 3 years of age

and the youngest 15 months. She nursed both of her children for ten to twelve months, chiefly on the left breast, the first child entirely so, as the right breast suppurated for two or three weeks. While nursing her first baby she observed a small hard lump at the outside of her left breast, and as it was painless and did not increase in size she took no further notice of it. It was only when her second baby was born twenty months later that she became aware it was increasing. She nursed the child on both breasts notwithstanding, and it was not for ten months, by which time the tumour had grown a good deal, that she weaned the child and sought advice at the Glasgow Royal Infirmary. In January of 1895 she was admitted to that Institution, and the journal report states that an examination shows the left mammary gland to be a little more swollen than the right one, and to present a hard and nodular appearance. In its centre is felt a large mass, measuring 5 in. across and  $3\frac{1}{2}$  in. in vertical diameter, while small nodules from this infiltrate the skin around. About 2 in. upwards and to the left of the nipple is seen an ulcer 1 in. in size, two nodules about the size of beans bordering on the extreme left of this ulcer. Patient appears to be strong, healthy, active, and robust.

On 25th January 1895 she was operated on. The Hospital journal says that the left breast was excised, a large area of skin free of tumour being taken away. The axillary glands were removed, also a considerable part of the pectoral muscle which appeared to be implicated. A plastic incision was made parallel to the trunk to allow of the edges of the wound being approximated. Patient seems to have made a good recovery, and to have left the Infirmary towards the beginning of March with the wound almost healed. About a month after she had gone home, that is, within three months of the operation, she noticed that the wound had opened, that a little discharge was coming away, and that pain of a shooting character had developed. She observed, also, that a hardness was growing at the side of the scar, and so she returned to the Infirmary for advice. She was there told that she should come into the Hospital again. She was readmitted for a few days and then discharged, as it was thought that an operation would be useless. The journal report is as follows:—

“28th April 1895.—Dismissed. General involvement of whole scar by large tumours, cancerous in nature, to remove which entirely was thought impossible. Adherent axilla and chest walls. One of the wounds from the recurring secondary tumours has given way, and there is now an ulcerated surface.”

Such, briefly, was the outline of her personal history as detailed to me. On questioning her, nothing could be elicited in her family history that showed any hereditary tendency to cancerous disease. On 11th May, at the time she presented herself to me, the local condition for which she sought advice was as follows:—

On the left side of the thorax there is seen a very extensive



cicatrix in the situation of the left mamma, which has apparently been entirely removed. The scar extends from the middle of the axilla to within  $1\frac{1}{2}$  in. of the xiphi-sternum. It is irregularly curved in aspect. Above the centre of scar is a cicatrizing area, which had broken out after the operation in January last. This is now granulating and seems healthy, but immediately below, and arching over centre of the long scar, is a mass of recurrent tumour, hard and nodular, with much thinning and discoloration of skin. This mass is curved in shape, about  $2\frac{1}{2}$  in. broad at its broadest part, and about  $3\frac{1}{2}$  in. in length. There are other nodules in the cicatrix as far back as the axilla. Four inches lower down there is the linear cicatrix of a plastic operation, made apparently to allow of the sliding together of the edges of the operation wound. No enlarged glands felt in axilla or above clavicle, but there is a distinct tumour of the left lobe of the thyroid gland, with some enlargement of the isthmus. This, however, she said had been present as long as she can remember. Right breast and axilla were free of any disease. Patient's weight was 9 st. 9 lbs. She looked pale and careworn, and when questioned admitted she felt ill, and was quite unable for her household duties.

From the clinical history she had given me, and from the local condition present, I had no doubt that the case was one of carcinoma, a diagnosis that was subsequently confirmed by our pathologist, Dr R. M. Buchanan, who reported as follows on a portion of tissue taken from the ulcerated surface above the line of the cicatrix:—

“The portion of tissue from the case of Mrs B. is typically cancerous. The cellular elements predominate over the stroma very largely.”

The question that had to be decided was whether anything further could be done for the case. As regards local removal, I was quite at one with the opinion already expressed at the Royal Infirmary that it was unjustifiable, because the prospects of complete eradication of the cancerous material were not good, and previous experience had shown me that in young patients, such as the present, the attempt is seldom successful, and, indeed, sometimes seems to hasten the progress of the disease, which assumes an acute and fulminating form, most disappointing and disastrous. Failing, then, local measures, could the disease be attacked in any other way, and by any other channels? To answer this last, it is necessary, Mr President, that I should put before you views that I have for some time held as to the etiology or cause of cancer generally, but more particularly of that of the female mamma. Before, however, doing so, I think it will be advantageous that I should very briefly lay down what I consider is the present state of our knowledge of carcinoma or cancer, so that I may make it quite clear what I mean by that term, and that there may be no difference of opinion as to what it is we are discussing. Well, I

think I put the case fairly when I say that there are certain points in carcinoma on which we are all agreed, and others on which there is great diversity of opinion. I think we are all at one on the following:—

1. That carcinoma is a tumour taking origin in epithelium, and having an epithelial structure.

2. That the essential feature of the disease is the continuous and excessive growth of this epithelium, which invades the surrounding tissues, spreads along the lymphatic vessels, passes from one set of glands to another, and eventually forms deposits in distant organs and parts of the body.

3. That once this proliferation of epithelium has begun, nothing that we know of has the power of arresting it.

4. That if a microscopic section of a carcinoma is made sufficiently thin and stained, certain special cells are observed, which cells, although not fulfilling the rôle of Lebert's specific cancer-cell, are yet sufficiently characteristic of the disease, and are now known as "cancer-bodies."

5. That clinically it is a matter of common observation that the younger the patient the more rapid the cell proliferation, and the more quickly fatal the disease; while in many old persons cancer assumes the atrophic or withering form from fatty degeneration and absorption of the epithelial cells, little more being left than a mass of fibrous tissue, with here and there a few cells surrounded by granular debris.

6. That cancer kills either by septicæmia from absorption of unhealthy products, or by hæmorrhage, or by interference with the function of some important organ.

7. That in our present state of knowledge of the nature and etiology of cancer, that the best treatment we can offer our patients is the complete removal of the disease by the surgeon's knife, and that the aseptic surgery of the present day allows this to be done more freely than heretofore, so that very extensive operations are performed nowadays.

There is, however, not the same unanimity of opinion on the two following points in connexion with cancer:—

1. As to the purely local origin of the disease.

2. As to the interpretation to be put upon the structures known as cancer-bodies.

Taking the first point, we find that some hold that the carcinomatous growth has a purely *local* origin—starts, in fact, from an irritation developed locally, and that, if that irritation and its effects are freely removed, the patient is cured (Hutchison). Others, again, teach that carcinoma, though an affection of the solid tissues, as shown in the local cell proliferation it causes, is really a blood disease, and that the tumour is only a local manifestation of a blood affection (De Morgan). Lastly, there is what I may term a third school, who hold that there is a certain state of the system or

Fig. 1.

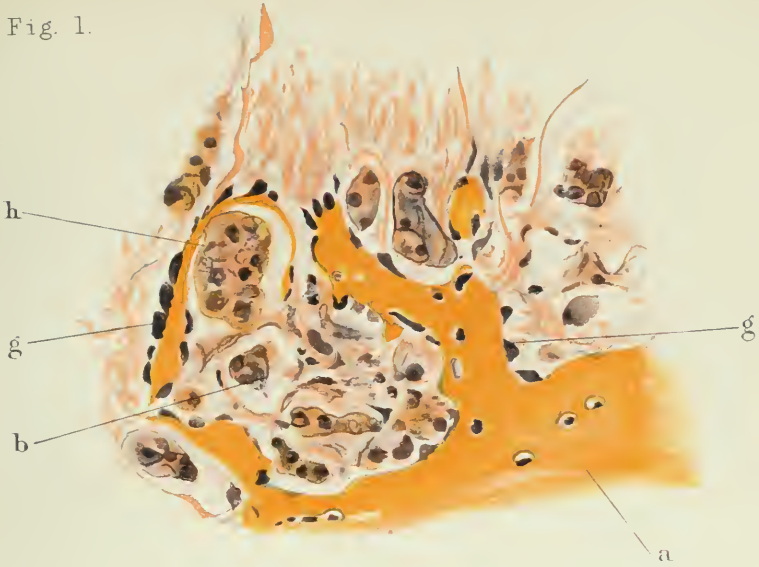
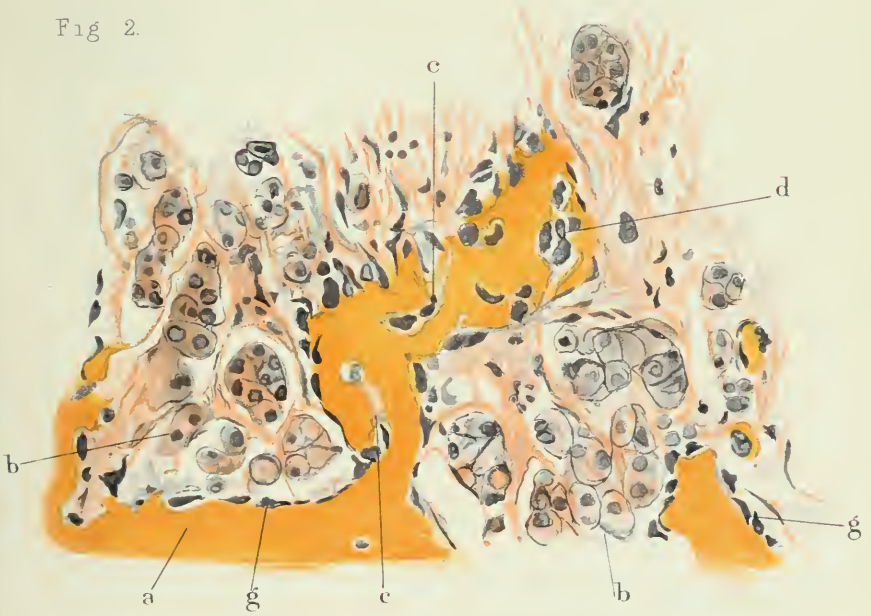


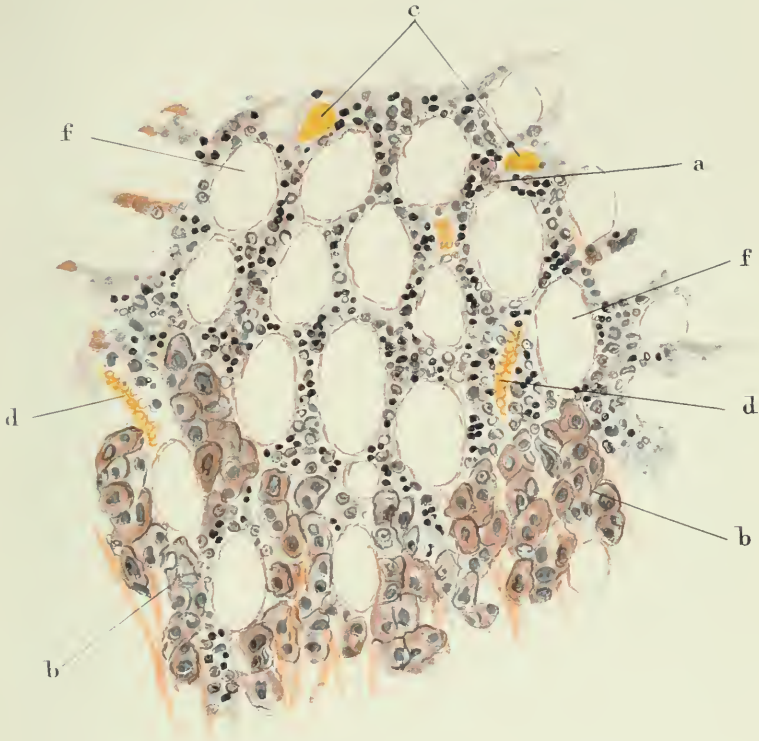
Fig 2.



SECONDARY CARCINOMA OF BONE.

a. Thin remains of bone. b. Cancer cells in columns. c. Bone corpuscles in tunnels which open towards the medullary cavity. d. Several bone corpuscles in one cavity. g. Bone corpuscles liberated and lying on inner surface of bone. h. Cancer cells in actual contact with bone.





SECONDARY CARCINOMA OF BONE.

MEDULLA. a. Medulla apparently healthy. (The greater part of the medulla seemed to be healthy).  
b. Columns of cancer cells invading the medulla. c. Spicules of bone. d. Bloodvessels. f. Fat spaces.



of the tissues in which a local injury, such as a blow, will start a carcinoma of the part, and without this local irritation a cancer will not develop (Paget).

Coming next to the interpretation to be put upon the cancer-bodies, a large number of observers, and amongst them men of the highest standing, look upon them as inter-cellular organisms of the nature of coccidia, or psorosperms, as French authors call them, and they regard them as the cause of the cell activity and proliferation characteristic of cancer. One distinguished member of this Society, Dr Russell, has brought out the fact that these cancer-bodies can be particularly well displayed by fuchsin staining, but, if I remember correctly, he looks on them as closely related to the yeasts. Others, however, are not satisfied as to the parasitic nature of these cancer-bodies. They explain them as arising from the embedding of leucocytes within certain of the cells, or, as Klebs puts it, from the fructifying influence of the leucocytes upon them; while others, again, think that they are simply epithelial cells undergoing vacuolation in the course of what is evidently a mucoid degeneration. I confess that of late this latter has been my own feeling.

I must now ask you to allow me briefly to mention to you what has led me to modify still further my views about these cancer-bodies, and also to lean to an explanation of the exciting cause of cancer that is quite opposed to the parasitic theory of the disease. I shall do so as shortly as I can.

It is just twenty years ago that I was asked to take medical charge of a gentleman whose mind was affected, and I went to reside with him at one of his estates in the West of Scotland. My duties were at times exciting, but never onerous, and I had a good deal of leisure to myself. I thought it would be a good opportunity of writing my M.D. thesis, and, after consideration, I decided I would take up the subject of lactation. What suggested it to me was the weaning of the lambs on a large adjoining sheep farm soon after I went down to my patient. Accordingly, I commenced to work at it, getting all the practical information I could about it from the farmers and shepherds round. At that time, however (1876), cerebral localization was being much talked about, and I took up the disease sturdy in sheep instead, as there were a good many cases of it just then. I yet, however, elicited the following points in connexion with lactation that struck me as of great interest:—

1. I found that the secretion of milk, though undoubtedly affected by the general nervous system, had no special nerve supply of its own to control it. Neither section of the sympathetic nor of the spinal nerves seem to influence it. The erectility of the nipple is affected by cutting the latter, but nothing more.

2. It was clear to me that the changes that take place in the mammary gland in the process of lactation are almost identical, up

to a certain point, with what takes place in a cancerous mamma. We have, under both these conditions, the same proliferation of generations of epithelial cells which block the ducts and fill the acini of the gland, but in the case of lactation they rapidly vacuolate, undergo fatty degeneration, and form milk, while in the carcinoma they stop short of that process, and, to make room for themselves, they penetrate the walls of the ducts and the acini, and invade the surrounding tissues. In short, lactation is at one point perilously near becoming a cancerous process if it is at all arrested.

3. I learnt this very remarkable fact, that it is the custom in certain countries to remove the ovaries of the cow after calving if it is wished to keep up the supply of milk, and that if this is done the cow will go on giving milk indefinitely. This fact seemed to me of great interest, for it pointed to one organ holding the control over the secretion of another and separate organ, and thus explained the absence of that distinct nervous control that I pointed out as characteristic of the mamma. Of course, the close intimacy between the ovary and the mamma is well known to all of us, as seen in the absence, as a rule, of the menstrual function during lactation, but I certainly was not aware until then that it was of the nature that it would seem to be, and almost of a distinct control. In our country farmers have not gone the length of spaying cows, as in Australia, but they attain the same end of having a continuous supply of milk by getting rid of all ovarian influence in another way. We know that during pregnancy the ovary is, as a rule, functionless,—that is to say, we have not the indications of its activity in the shape of the menses, and it would seem to be in its turn brought under the control of the pregnant uterus. Farmers knew that their cows after calving usually begin to menstruate every three weeks, and that with the establishment of this function the mammary secretion gradually lessened. They also knew that during the nine months the cow carries her calf she did not menstruate, so to prevent menstruation and lessened milk they put the bull to the cow usually two or three months after the calf is born and when the milk secretion is becoming lessened, the result being that with pregnancy the secretion ceases to lessen and remains copious.

I need hardly say that, though I temporarily abandoned the subject of lactation for my thesis, I did not lose sight of the facts above mentioned, for they seemed to me to point to influences at work in the human system that had not as yet been generally reckoned with or recognised. Above all, I was struck with the local proliferation of epithelium seen in lactation. Here was the very thing characteristic of carcinoma of the breast, and, indeed, of the cancerous process everywhere, but differing from it in that it was held in control by another organ, and could either be arrested by that organ altogether or continued to a further stage, where



the cells became fatty, and passed out of the system not only in an innocuous but nourishing fluid—milk.

Now, gentlemen, I think I am correct in saying that the spirit of modern pathology is this,—that all pathological changes are merely modified physiological ones, that there is no essential difference between the two, and that a knowledge of the forces controlling the one may sometimes give us a clue to the other. I often asked myself,—Is cancer of the mamma due to some ovarian irritation, as from some defective steps in the cycle of ovarian changes; and if so, would the cell proliferation be brought to a standstill, or would the cells go on to the fatty degeneration seen in lactation were the ovaries to be removed? For an answer to these questions I felt I must wait; but, on settling in practice in Glasgow in 1878, I determined to look further into this point of the control the ovaries seemed to have over the function of lactation. Accordingly, I obtained at the end of 1878 a licence for performing the experiment of removing the ovaries from suckling rabbits. Through the kindness of Prof. M'Kendrick, I was able to carry my experiments out at the University laboratory. Time will not allow me to go into them in detail, but I may say that the three cases I tried all confirmed the fact. As long as the young ones were at the breast the milk supply continued, and when eventually they were taken away the milk supply ceased; but the creatures increased very much in size, and post-mortem examination revealed that this was due to large deposits of fat around the various organs, and above all in the lumbar region, where there were masses of pure adipose tissue, showing that the secretion of milk was still going on, but, not being discharged by the usual channels, was deposited in the various tissues of the body as fat.

In the year 1882 a case of uterine cancer, unsuitable for local removal, came under my care, and I thought I would try on it the effect of removal of the tubes and ovaries, as she was willing to submit to any operation. I found, however, on performing abdominal section that the disease had extended so much into the broad ligaments that a satisfactory removal of the appendages could not be accomplished, and I abandoned the operation. She made a good recovery from the laparotomy, so that no harm was done by it; and she died some months later, I was told. With this single attempt to put my views to the test I was for a time content, as I was very unwilling to do anything of the nature of experiments on my fellow-creatures. Further, with the rise and progress of bacteriology, I began to share in the hope that in this quarter a solution of the true nature of cancer would be found; and, with the announcement of the so-called cancer-bodies now generally recognised, I began to think less and less of my ovarian theory of the origin of cancer.

On taking up my work at the Glasgow Cancer Hospital, which I may say has been established not only for the treatment of

cancer in all its stages, but also for the pathological study of the disease, I felt that the position of matters was that our present state of knowledge has nothing better to offer than the surgeon's knife for the cases where the tumour was limited and could be thoroughly removed; but that in inoperable cases, if the so-called cancer-bodies were not parasites at all, but merely cells undergoing mucoid degeneration, it was possible a free administration of thyroid extract might influence the growth, and work through time a cure. Failing this, I thought I might follow up my old line of reasoning, and in cases of advanced carcinoma of the breast in young patients see what effects the removal of the tubes and ovaries would have on the progress of the cancerous growth in the way of arresting the cell proliferation and converting the cells into fatty matter.

Although the breast had been removed, this was the line of procedure I decided to adopt with Mrs B., and accordingly on 11th May she was put upon the thyroid tabloids. They were pushed until their physiological action was made apparent; but, as no appreciable change was seen in the diseased condition at the end of a month, I put it to her husband and herself as to whether she should have done the operation of removal of the tubes and ovaries. Its nature was fully explained to them both, and also that it was a purely experimental one, but that it could be done without risk to life; and that, if it should have no effect on the cancerous process, it would cause her no increase of suffering. She readily consented that I should do anything that held out any prospect of cure, as she knew and felt her case was hopeless. On 15th June I operated, and removed the tubes and ovaries on both sides. The right ovary seemed healthy; the left one was somewhat cystic. Subsequently there was some little trouble with the action of her bowels; but she made a good recovery, and on 28th June was sitting up. *No local application was made to the diseased areas on the thorax. They were simply kept clean with boric lotion, and dressed with protective and borie lint.*

On 12th July the administration of the thyroid tabloids, three daily, was resumed, as I felt that, though I hoped by my oöphorectomy to arrest the cell proliferation and favour perhaps fatty degeneration of the cells, there was present such a large amount of cancerous material that a powerful lymphatic stimulant such as thyroid extract might be useful.

On 19th July, about five weeks after operation, an examination of the diseased areas on the left side of thorax showed, as the Hospital report states, undoubtedly a marked change compared with their condition some weeks ago. The larger mass of disease is much less vascular. It is also smaller, flatter, and altogether less prominent, and the same may be said of all the other secondary foci of disease. The tissues around are also softer and more pliable.

On 1st August it is noted that the local improvement continues, and that the measurements of the largest area of disease are—length,  $2\frac{3}{4}$  in.; breadth,  $1\frac{1}{4}$  in.; while the depth is hardly appreciable. The colour is a dull yellowish-white, and the vascularity slight. There are five small nodules in the axillary region, which are also diminishing in size and vascularity, though perhaps not so much as the larger growth. Patient's general health and nourishment are satisfactory; and, as she is an intelligent and reliable woman, and interested in her own case, I allowed her to go to Bridge of Allan for a change, and asked her to report herself from time to time. This she did, and, without going into a detailed account of her condition on each visit that she made, I may say that the local improvement continued, and my note on 12th October, just four months after the operation of oöphorectomy, was as follows:—"On examination of the left breast the condition of the tissues is favourable. The most remarkable feature of the case is the yellow fatty look that the former thick bar of cancerous tissue above the scar of the incision for removal of the breast presents. It is to my mind the most striking feature of the case. The cancerous tissue has been reduced to a very thin layer, and is in no way raised above the surrounding skin. In fact, the whole surface is smooth and level, and to the naked eye it seems as if the skin at this part had a yellow look. So distinct is this that one could easily trace out the outline of this yellow-coloured tissue. At places the surrounding skin seems pushing its way into the yellow mass, and the processes of bluish cicatricial tissue are to be noted. The yellowish nodules at the axillary end of the incision are still apparent from their colour, but they seem thinning out. The whole of the tissues on the chest wall are more movable, and the surrounding skin has a clear and healthy look. The scar of the former ulcer above the mammary excision cicatrix is sound, and no new nodules are at present observable. Patient expresses herself as feeling very well, and looks so. She is taking four 5-gr. tabloids of thyroid extract daily."

I need not trouble you with any further detailed account of this patient than to say that eight months after my operation all vestiges of her previous cancerous disease had disappeared, and that I am able to show her to you to-night with a sound cicatrix and healthy thoracic tissues, and that she is apparently in excellent health.

The next case that I wish to bring under your notice is that of Margaret R., *æt.* 40, married, no family, who was admitted to the Glasgow Cancer Hospital on 2nd September 1895, suffering from a large tumour of right mamma. It had existed to patient's knowledge for five and a half years, and had not been operated on. Her family history was satisfactory, and personally she had

been a very healthy woman all her life. She began to menstruate at 13 years of age, and the menses still continue. At present they come every three weeks.

The account she gave of the appearance of the tumour was that it followed an injury five and a half years ago, that at first it grew very slowly, but that it has increased much more rapidly since she sustained a blow on it from a door nine months ago. Further, she has had continual pain in it as well as in the neck, the pain passing through to the back. It comes on more severely at times, and is of a shooting nature, and at nights keeps her from sleeping by its severity. She has got thinner, but has not felt specially ill or weak.

Her condition on admission, as noted in the Hospital journal, is as follows:—She is pale and worn-looking, and gives the impression of being a nervous and emotional woman. Locally the right breast is occupied by a large tumour, which involves the whole organ, save its extreme lower margin. The tumour is densely hard, but uniformly so, and more or less rounded and smooth. It is adherent to the skin over a large area, this skin being infiltrated and reddened. The mass is somewhat tender. At its upper part there is a distinct cutaneous nodule about half an inch long and one-fifth of an inch broad. The tumour is not fixed below, though there is no great freedom of movement. The skin around the breast, especially at its lower part, shows congested vessels, and is apparently hyperæmic. The nipple is retracted and fixed. In the axilla there are a number of enlarged hard glands rather deep and fixed. The pectoral fold running up to axilla is slightly thickened and indurated. In first interspace some small shot-like nodules are felt under the skin. Also over the clavicle under the skin there are quite a number of them, and they give a rough feeling to the surface of the bone. In the supra-clavicular space, and in the posterior triangle of the neck, there is a wide infection of the lymphatic glands—one especially hard and fixed mass lying slightly above the clavicle. Numerous enlarged glands can be traced as high as the lobe of the right ear. On left side, in anterior triangle of neck, just above inner end of clavicle, there is a small, hard, enlarged gland. There is no œdema of arm, but patient complains of almost constant pain down each side of the arm, and of pain in the neck. Left breast and axilla seem normal. No secondary deposits can be detected in any of the organs of the body. The only suspicious point is that the percussion-note over and just below the right clavicle is impaired, while, anteriorly and posteriorly, the R.M. is somewhat prolonged and hard at apex, and a slight creaking and grating can be heard. Otherwise the lungs are normal.

I learnt from her that she had recently applied for admission to one of our local infirmaries, but had been refused, as the surgeons who saw her considered that nothing in the way of

operation could be done. I was of the same opinion, for the disease was evidently carcinoma, an opinion subsequently verified by Dr Buchanan microscopically, and any attempt to locally remove the disease would be futile, I considered; but after what I had observed in the previous case related to you, I was not without hope that something might be done to retard the progress of the disease, although, from its great extent, as compared with Mrs B.'s case, I was not so sanguine of success. However, I admitted her to the Hospital. What I looked on as the unfavourable feature of the case was the implication of the glands, not only in the neck, where there was a hard and irregular chain of them, matted together and quite fixed, running up the posterior border of the right sterno-mastoid, but also in the axilla, where there was a deep and immovable mass of them. After admission she had to have half-grain pills of opium for the pain, which seemed severe. I put before her the question of the removal of the tubes and ovaries in her case, but she could not bring herself to submit to an operation which I could advise her to undergo as regards the risk involved in it, though not able to promise her with certainty a successful result in the matter of cure. Another point that made me not very keen in urging the operation was that I found she was a woman of highly nervous temperament, and rather addicted to alcoholic excess. After her admission, quite a month elapsed without anything being done. During that time it was clear that the disease was extending, as the journal of 30th September says—"Extension of disease is seemingly rapid"; and again on 2nd October it is noted that "the skin over the prominent nodule above the centre of the upper margin of the tumour is reddened, and that downwards and inwards, diagonally from the nipple, there is another nodule of smaller size; while on outer side of breast, on and beyond the margin of the tumour, lying in an area of congested skin, there are nine separate and distinct small nodules in and just under the skin, the size of peas or small shot. In the right axilla were three cutaneous nodules, and the deep mass of hard fixed glands; while below the centre of the right clavicle was an irregular mass of enlarged glands evidently growing quickly. Above the clavicle in the anterior and posterior triangles of the neck are the glands much enlarged and matted together, and extending in front of and behind the sterno-mastoid as high as the angle of the jaw. On the left side of neck there is present a small adherent nodule between the tendons of insertion of the left sterno-mastoid and an enlarged left supra-clavicular gland."

At this time she felt she was not improving, and consented to my doing anything that I thought would benefit her condition, so, on 3rd October, I removed her tubes and ovaries. The operation was not an easy one, as the uterine annexa were very adherent, thickened, and altered in appearance as well as in situation, the ordinary anatomical arrangements of the parts being quite

obscured. The after-progress of the case was, however, uninterrupted, and at the end of three weeks she has completely recovered. The journal note of 12th October says that it is evident that the cutaneous surface of the tumour is less vascular, the red blush which covered it entirely being very noticeably diminished, while on 14th October it is remarked that to-day patient states very emphatically, of her own accord, that "she feels in a different world," the pain in her breast being so much easier as to be almost gone. She says, too, that she moves her neck more freely and with less pain. In confirmation of the diminished pain, the matron stated that she slept well at night, and had not required any opium since the operation. On 14th October she commenced to take two 5-gr. thyroid tabloids twice daily, as I decided to follow strictly the mode of procedure in Mrs B.'s case. On 9th November, five weeks after the operation, it is noted that the mass above the clavicle certainly seems smaller and less fixed, the neck being now moved freely in any direction. Diminished size and vascularity in the breast tumour are admitted by all who have watched the case to be apparent, though slightly marked, while the nodule at upper zone of breast is superficially distinctly less, and has a yellowish dull colour, quite different from its former glazed and red appearance. On 3rd December, two months after operation, the above nodule, as it continued to show decided atrophic changes, and to shrink, was removed under cocaine, so that it might be examined by the pathologist to the Hospital, Dr R. M. Buchanan. The journal report continues:—"There is now a striking difference in the size of the tumour of the right mamma as compared with its former state, and over the inner and upper parts of the tumour the skin is becoming freer, and can now be pinched up, though it is still distinctly thickened and infiltrated. Formerly it was quite impossible to grasp it at all, so firmly was it adherent to the tumour." The patient's progress was unfortunately interrupted by a septic suppuration which followed the excision of the nodule under the cocaine, and on 9th December I had to open an abscess over the shoulder, the scar of which can still be seen anteriorly. After this she got on well, and the spot where the nodule was excised cicatrized soundly, and can now be seen quite healed. This I thought interesting, as cutting out a nodule from a cancerous breast is very apt to leave a sore that refuses to heal, and even fungates. The report, from the pathologist, of the microscopic appearances of the excised nodule was that there was a great increase in the stroma, and that the epithelial cells were undergoing marked fatty degeneration—in other words, that there was a more cicatricial condition of the tissue, such as we see in the cases of atrophic or withering scirrhus mammae.

On 10th January Mrs R. was allowed to leave the Hospital to go to the coast for a change. I am afraid that while absent

she did not take that care of her health that I should like, and was remiss in taking her tonic and her thyroid tabloids, and on her return on 25th February, although looking well in herself, I did not consider the local condition so satisfactory, the glands in the neck being more attached to the skin, larger, redder, and more fixed. Over clavicle an increase of infiltration is occurring, while in axilla some fresh small nodules have arisen. The breast itself seems more adherent again to the skin, and redder, and the outlying nodules larger.

On 2nd March I readmitted her to the Hospital, as I was satisfied she was not doing justice to herself outside, and was not allowing the remedial measures that had been employed to have a fair chance. She was kept in bed, and three thyroid tabloids administered daily. Under these measures she improved gradually and seemed to be free of pain, sleeping well without any opiates.

On 7th April there is noted the increased size of the nipple of the right breast. It is of a pale pinkish colour, somewhat glossy, is like a raspberry in shape, and about four times the size of the left nipple. It now stands out prominently from the centre of the tumour, while formerly it was sunken and retracted.

I have brought this patient before you to-night, not putting her forward as a case that is cured, but simply as illustrative of the changes that were noted in Mrs B.'s case, but have now entirely disappeared in her. Thus, in the enlarged supra-clavicular glands you will, if you examine them closely, note the areas of yellowish-white coloration, which indicate, I consider, the presence of fatty degeneration, a condition present in the cancerous masses in Mrs B.'s case before they eventually disappeared. The same can be seen in several of the nodules in the vicinity of the breast, and recently one of them, just above and to the right of the nipple, broke, and has left a yellowish scab with a subjacent ulcer. A comparison of the two casts of the right mamma taken at intervals of five months shows the difference in the size of the mammary tumour at the two periods. Altogether, I am inclined to think that the disease is in a more quiescent stage, and gives some indications of a possible cure.

The last case I wish to bring under your notice is that of Margaret M., æt. 49, unmarried, who was admitted to the Glasgow Cancer Hospital on 2nd December 1895. She sought advice for a large sore of the left breast, but, as you will see, the left breast has been entirely eaten away, almost as if it had been removed by operation, but she assures me that nothing was ever done to it, and that she had had no advice about it until she came to the Hospital. The disease seems to have existed for six or seven years, and to me one of the remarkable features about it is that it has remained so localized, none of the adjoining glands being apparently as yet involved, and no evidence of any secondary

deposits in any organs of the body. I was at first inclined to think it was possibly a tubercular ulcer we had to deal with, but the removal of a portion of tissue from the margin and its examination by the pathologist showed it to be "typically cancerous." The report adds, "There is no evidence of tuberculosis."

As the menopause had occurred with her two years previously, I was not prepared to at once remove the tubes and ovaries, and thought it might be a case where the thyroid tabloids might be alone administered with the view of seeing what effect they would produce, and whether under their administration any of the vascular diminution and atrophic changes seen in the other cases would be seen. Accordingly, the sore was got into as healthy a condition as possible, and on 7th February, when it was clean in its deeper parts, and even showed at places some attempts at cicatrization, the administration of the thyroid tabloids was commenced, and they have been continued since, being pushed to their full physiological effect. During the three months they have been taken I cannot say that I observe any marked effect on the sore. The indurated and infiltrated borders are apparently unaltered, and show no diminished vascularity, while the ulcerated area has increased below and seems extending. By itself thyroid extract seems to have little effect on the cancerous process, and the question I consider that is opened up by this case is, whether in the light of what has been observed in the other cases I should see whether the removal of the tubes and ovaries in this case would be of any service. I am satisfied that it would be a mistake to attempt the local removal of the sore. I am not so sure, if my view that ovarian irritation may be the exciting cause of cancer is correct, whether it would not be right, even though the menopause has set in, to see what effect the removal of the uterine appendages would have. If this is done, and the Society desires it, I will gladly read a note of the case at some future meeting and show the patient.

Gentlemen, although I have already trespassed very considerably on your time, I am going to ask you to bear with me while I place before you what I believe to be the interpretation of the changes seen in the two cases I have brought under your notice this evening. The conclusion I draw from them is this, *that we must look in the female to the ovaries as the seat of the exciting cause of carcinoma*, certainly of the mamma, in all probability of the female generative organs generally, and possibly of the rest of the body. I have felt for some time that the parasitic theory of cancer is an unsatisfactory one in many ways, and that in directing all our energies to working it out we are losing time and searching for what will never be found, simply because it does not exist.

Further, and bearing on this point, I think we are perhaps in error in assigning to the nervous system the entire regulation of



the metabolic changes in the tissues of the body. I am satisfied that in the ovary of the female and the testicle of the male we have organs that send out influences, more subtle it may be and more mysterious than those emanating from the nervous system, but possibly much more potent than the latter for good or ill as regards the nutrition of the body, and, undoubtedly, whatever cancer may be, it is very generally admitted that it is a disease of the nutrition of the part affected.

To make clear to you how I think the ovaries may be the exciting cause of carcinoma in the female, I must ask you for a moment or two to consider reproduction as it takes place in the lowest animals and in the human body. In the former we have no special cells or organs for reproduction. If a hydra is cut into a number of portions, each portion will develop into a separate and complete hydra, as Trembley showed more than a hundred years ago. This would seem to indicate that all the cells of the creature possess equally the reproductive power. As we get higher in the scale of life, the principle of *division of labour* comes into play, and in the human body there is seen at an early stage of development the arrangement of the cells of the body into separate layers, and subsequently the differentiation or specialization of these cells into separate organs and tissues. In other words, these cells become of a higher type. But there is one very definitely marked-off group of cells which retain their primitive condition, and do not specialize into a higher kind of cell—I refer to those whose function it is to reproduce the species. In no way dissimilar at first, they are separated off to form either ovary or testicle as the case may be, and through their agency the continuation of the race is assured. In the ovary we have well demonstrated the active proliferating power of these cells, for from it are derived several epithelial formations—viz., the surface epithelium of the ovary, the epithelium of the Graafian follicles, and the cells of the ovary that we speak of as ova.

Now, I consider, comes in this very important question—When these reproductive cells are set aside for this one function, do they take away from all the other cells of the body their reproductive power, leaving them only the ability to form the special tissue for which they are intended, or do these cells still retain their reproductive power, but have it kept in check and control by the ovaries as long as these organs are healthy? I am inclined to the latter view, though I am aware that it is not in harmony with Weismann's teaching on heredity, which would have us believe that there is a very definite distinction between the *somatic* and *germ* cells, the latter being handed down in unbroken continuity from parents to offspring, and having nothing whatever in common with the somatic cells, they simply having, as it has been very well put, only board and lodging in the human body. I have never felt sure of Weismann's theory. It is no doubt ingenious

and attractive; at the same time it is quite possible that this doctrine of absolute continuity of germ plasma may be incorrect, and, if at variance with what is observed in disease, it shows it to be faulty in a very important respect.

Going, then, on the assumption that the ordinary cells of the body have not lost their reproductive force, but that this latter is held under control by the healthy ovaries, which are simply masses of germinal epithelium, I can conceive it quite possible that any altered secretion of these organs, or any morbid condition of them, might so affect the other cells of the body as to allow their latent reproductive power to come into play, and thus confer on these cells the active proliferating powers of the germinal epithelium. And this is what at present I am inclined to think does take place, and that cancer consists in the epithelium of the part affected taking on the active proliferation which is the marked characteristic of the *germinal epithelium*; and, though I am not yet in a position to assert it as demonstrated absolutely, I have the belief that the special cells seen in sections of cancer, and known as cancer-bodies, will eventually be shown to be special germinal cells corresponding to the ovum cells elaborated by the ovary.

After this idea had occurred to me, I was interested to find that Klebs has come to the conclusion that in cancer epithelial cells become transformed into ovum cells. He attributes this to the leucocytes exerting a fructifying influence upon the cancer cells and causing them to multiply. I, on the other hand, while agreeing with him as to this fact, think that in the female there is some ovarian influence which works the change. It may be an altered secretion, or it may be the migration of cells, it might even be a parasite in the ovarian cells, for it should be borne in mind, in regard to the secretions of the reproductive glands, "that, unlike other secretions, their essential constituents are living cells" (Stewart), but in whatever way brought about, there seems to me a reasonable ground for thinking that the active processes seen in a cancerous tumour are best explained by regarding the epithelium of the part as having taken on the properties and powers of the *germinal epithelium*.

It may be said that in many cases of cancerous disease no changes are perceptible in the ovaries. This may be so, because they may not have been looked for and because they may be of a delicate nature and requiring special investigation. We know that there are certain coarse lesions of the ovary that certainly are of a malignant and infective kind, and are typical of what we meet with in cancer. I refer to those cystomata of the ovary which sometimes burst and infect the whole peritoneum, and even the abdominal organs themselves. I have operated on such cases and had to close the abdomen, as it would have been useless to attempt removal of all the infected parts. I was struck by a

paper published by Professor Alex. Simpson, in one of the recent volumes of the *Edinburgh Hospital Reports*, where such a case had occurred to him, and in the remarks he made upon it he formulates the view that removal of both ovaries seems to predispose to cancer. If this were so, then my theory to explain my success in Mrs B.'s case must fall to the ground, but the point that Professor Simpson has overlooked is that in all the cases he refers to the ovaries were removed *because they were diseased*, and thus these cases in reality support my view that cancer in the female is in all probability due to some altered ovarian condition, which may be either a coarse lesion of the organ or something more hidden or obscure, such as an altered condition of the cells and their secretion. If cases can be shown that have had both healthy ovaries removed and have subsequently been the subject of cancer, then my theory falls to the ground.

Further, if the view I put forward is a correct one, then cancer in the male should be due to some altered condition or secretion of the testicle, for this organ is built up of cells that, at one stage of their existence in early foetal life, are so identical with those that form the ovary, that for some weeks they are not distinguishable one from the other. Well, I have been looking into this matter, and one or two facts of considerable interest have come to my knowledge, showing that the testicle seems to have the same control over local proliferation of epithelial cells, such as is seen in the ovary and lactation. Thus, in stags the yearly growth of the horns, which is a local cell proliferation, is under testicular influence, for if a deer is castrated its horns do not grow; and what is more remarkable still, if only one testicle is taken away it is only the horn on that side that does not grow. I have arranged with a friend of mine to carry out this season some experiments on lambs, by castrating them only on one side, and to watch if the horn on that side is absent. Also, I am making inquiries as to the existence of cancer amongst eunuchs, for if my view is correct they should not suffer from it.

The only other explanation that might be urged in favour of the removal of the tubes and ovaries affecting the progress of carcinomatous growths, by those who hold the parasitic theory of the disease, is that the operation so profoundly alters the nutrition of the tissues that the parasite no longer finds a suitable nidus for its existence, and so perishes. I confess I think this is not likely, though it does not call in question the utility of the operation.

To recapitulate, I would urge the following points:—

1. That there seems evidence of the ovaries and testicle having control in the human body over local proliferations of epithelium.

2. That the removal of the tubes and ovaries has an effect on the local proliferation of epithelium which occurs in carcinoma of the mamma, and helps on the tendency carcinoma naturally has to fatty degeneration.

3. That this effect is best seen in cases of carcinoma in young people, a class of case where local removal of the disease is often unsatisfactory.

In conclusion, gentlemen, I would say that my paper has been written without the desire of being in any way dogmatic in the matter of the etiology of cancer. I desire it to be if anything suggestive. I am sure that many of you, like myself, while very desirous that a parasite should be found in cancer, as it would thereby render our chances of dealing with the disease more hopeful, have felt that this view does not satisfy the case, and leaves much to be explained. On the other hand, facts such as I have observed indicate, I think, that it is possibly in the direction of an altered condition of the ovary and testicle that we are to look for the real exciting cause of cancer, and if so, the sooner we direct our energies into that channel the better. I know that I have had nothing in the way of great results to show you to-night, but you must remember that I have worked with most unpromising cases, and that when you have present large masses of cancer it is not easy to bring healing influences to bear upon them. They have got beyond control, whereas in an early stage they might have been amenable to treatment. I am not standing here to-night advocating a wholesale removal of tubes and ovaries for carcinoma. My paper is headed "The Treatment of Inoperable Cases," and I am not in a position to ask you to replace the old plan of local removal—which, no doubt, brings us many disappointments, but has had of late years many encouraging results—by a new and untried method. All I feel is that there are grounds for the belief that the etiology of cancer lies, not in the parasitic view, but in an ovarian or testicular stimulus, and that the whole subject requires careful working out. I need hardly say that if that view is found correct it must materially modify our present lines of treatment.

I know it may be said that I am premature in bringing these cases before you, but I am well aware that in the field in which I am a humble worker there are others labouring as equally desirous as myself for the solution of this problem of the cause of cancer, and I am only too glad that they should know any facts that I have observed, and that may give a clue to the direction in which to work. I am also well aware that the views I have put forward to-night are to a large extent problematical; but whether you accept them or not, I am sure you will acquit me of having acted thoughtlessly or recklessly, and will believe that in all I have done I have had some reason for the faith that is in me, and that I have been actuated solely by the motives that guide all of us in the exercise of our profession,—primarily, the interests of those who place themselves under our care; and, secondarily, the progress and advancement of the healing art.

*The President* said he was sure the Society was deeply indebted to Dr Beatson for having brought before them these very interesting cases, and for his admirable exposition both of the actual cases and of the theory he had advanced in explanation of the grounds on which he had treated, and proposed to treat cancer, more particularly cancer of the mamma. There was no question that the operative treatment had had a marked influence on the progress of the diseased condition of the mamma. Whatever the nature of the change might be, the tumour had diminished most materially, and the patients' health improved. Whether these cases would command their belief in the great doctrine which he had brought forward, that cancerous affections might be due to some irritation from the sexual organs either in the female or in the male,—that cancerous conditions might in all cases be attributable to such a cause was perhaps, to say the least of it, doubtful. There were many irritative conditions of the generative organs which were not associated with cancerous affections of any part of the body, and the association with such conditions would require a large amount of further observation in order to be established. At the same time, he thought Dr Beatson was so far warranted, in that his idea was corroborated by recent investigations, which had shown that the nutritive conditions of the prostate, for example, were very materially influenced by the removal of the testes. That seemed to have been thoroughly proven; but it was a very wide and extensive theory which he had promulgated. He (the President) would be very glad to hear the observations of those able to speak with more authority than he could do on the nature of the theory and the facts which had enabled Dr Beatson to evolve it.

*Prof. Simpson* said that he agreed with the President that the Society was greatly indebted to Dr Beatson for the admirable paper he had brought before them to-night. They could all heartily endorse his closing sentences that they should regard it as a contribution made in good faith for the purpose of advancing science, and it deserved quite in an unusual way the expression of their thanks to-night. That was not to say, however, that he was prepared to adopt right off Dr Beatson's theory. Various points required explanation; *e.g.*, he told them he made experiments on rabbits, removing their ovaries, and finding that they got fat afterwards. Well, sometimes pigs when spayed might be fattened apart from lactation altogether. In the history of the female, when the ovaries began to lose their function a certain group of women had a tendency to *embonpoint*. That a deposit of fat took place had long been recognised, and that tendency asserted itself on the disappearance of the sexual function. Further, Dr Beatson had referred to his (Prof. Simpson's) paper, which had not quite regard to the point brought before them. His point was this:—That the patient who came under observation in the ward was judged to be subject to ovarian tumour. On opening the

abdomen they found two tumours. Both ovaries had undergone cystic degeneration. There was an unusually large flaccid wall in the case of the left. The liver had quite distinct cancerous nodules in it. The patient recovered from the operation, but died a few months later from the cancer that they had seen in the liver. He did not know if cancer had ever been seen before in that particular way. On looking into the relation of ovarian disease to cancer, one found that a considerable proportion of patients operated on for ovarian tumour had died afterwards from cancer. Spencer Wells had called attention to it. On looking into the matter lately more particularly, he found that, when both ovaries were removed, patients died in a considerable proportion of cases from cancer which developed sometimes in abdominal organs, sometimes in thoracic and other viscera. He did not remember the exact figures at present. But if, say,  $4\frac{1}{2}$  per cent. deaths of women in any year were from cancer, then some 28 per cent. of those from whom ovaries had been removed died of cancer. Therefore when the ovaries had been removed, especially both, there was a special proclivity to cancer. Olshausen and others supposed it due to engraftment of peritoneum with some epithelial cells left there from operation; or that perhaps the tumours themselves, supposed to be innocent at the time, really contained some malignant element or malignant proclivity. His (Prof. Simpson's) case seemed to show that the tumours might be perfectly innocent, because the diseased ovaries were carefully examined, and the degenerations were of the simple cystomatous character, but associated with carcinomatous degeneration in the liver, and that led him to make the suggestion that abeyance of function in the ovaries, still more the removal of them, gave proclivity to the development of cancer. Dr Beatson's paper to-night did not affect Sir Spencer Wells' statistics. He (Prof. Simpson) quite well believed that the ovarian function modified in quite a distinct fashion the tendency to change in the mamma, and the cases of Dr Beatson were of the highest interest, especially the first case; but they had to keep in view that the patient had had other treatment as well; and while the thyroid extract had value in many directions, it was quite possible that it might exert an influence in this case, perhaps all the more when the ovary had been removed. There were various elements in the system that required to be taken into consideration. The ovaries, he had no doubt, exerted part of their function by their influence on the nervous system, also partly by their chemistry, whatever that might be. In the *Centralblatt für Gynäkologie* a writer had recently drawn attention to cases where women had suffered from giddiness, flushings, and distresses of the climacteric. He first administered ovarian extract, ovarian tabloids in some form, and had got one of his assistants to make experiments in transplantation of ovaries from one side to the other in rabbits, engrafting in

peritoneum or in cellular tissues. The ovaries not only lived, but continued their functional activity. Their attention was thus called to the circumstance that the ovarian influence on the system—in this he hardly agreed with Dr Beatson—was not purely nervous, but was largely chemical, whatever the secretion or product might be. But that was a long way from saying that a change in ovarian function should start epithelial cells in other organs, even in an organ so closely allied to the ovary as the mamma, on a career of ovulation or development of ova. It was a kind of exaggeration, to say the least of it, to speak of cancer cells, although there was the high authority of Klebs for it, as being ova. He did not think any one would suggest that if conjunction could come about with spermatozoa from another individual that these would have any true ovular value whatever. At the same time they had a great deal to learn yet in regard to the development of cancer, and possibly also in regard to the development of embryonic structures generally. A very interesting discussion was in progress in a German Gynecological Association in regard to a matter discussed in London last week, of which they had seen no example in Edinburgh, viz., a deciduoma malignum, first described as a sarcomatous growth developed in decidual tissue; but some of his countrymen had alleged that this was a misnomer, and that it was not the decidua that underwent degeneration, but that it depended on some fragments of ova,—some villi of the chorion that remained in the uterus, which had gone on growing with a kind of independent growth, sometimes spreading into the vagina and going as far as liver and lungs; so that the kind of cells found in cancerous tissues ought not to be spoken of in such terms. At the same time the paper was one of great interest and would set other minds working, and they hoped that Dr Beatson would carry out his experiments. In that last case, *e.g.*, supposing the thyroid extract were mainly to receive the credit of it, it seemed to him a different variety of cancer. It was quite possible that thyroid extract might operate differently in different patients. In this case the woman was beyond the climacteric, a point to be carefully considered, because if it was a healthy ovarian action that was in some way stimulating the cancer growth—he understood that was the idea in Dr Beatson's paper—the fact remained that there was more tendency to the development of cancer after the ovaries had ceased functional activity.

*Dr W. T. Black* said that having once been a sanitary officer in the army he took a different view of cancer. From its extreme rarity in the army and navy, and among the Jews, who all have their butcher meat inspected before cooking, he deduced its prevalence in the civil population to the accidental use of infected meat. On inspecting the veterinary journals for some time back, he had found numerous cases related of malignant diseases in the domestic animals. In the human subject its

prevalence primarily along the digestive tube from mouth to rectum should lead to the same suggestion of its origin. Dr Beatson's view was highly interesting, being founded on physiological and pathological grounds. It was a hopeful paper.

*Prof. Chiene* remarked that he need not say he was very pleased indeed to rise and corroborate all that *Prof. Simpson* had said and what the President had said with reference to the value of this paper. It was valuable from the fact that Dr Beatson had been looking into this for twenty years before publishing anything upon it. He had taken care to go very slowly and gradually into it. As regards the theory, he (*Prof. Chiene*) was not going to enter into it. He could not follow it, could not understand it; but he would have them look at that preparation there, which was undoubtedly a carcinoma of the mamma, and he would say that every practical surgeon in the room, as he looked at that preparation and that woman there, was bound in inoperable cases of cancer to give a trial to removal of the ovaries along with the administration of thyroid extract, explaining to the patient what he was doing. There was a patient in his ward at present who had come from a distance wanting an operation. There was great pain and discomfort. The arm was swollen. The axillary glands were pressing on the vessels. The tumour was firmly attached to the chest wall. The difficulty in the case was that she was 40 years of age, still menstruating, but near the end of the menstrual period. Now, was he not justified in telling that woman what he had seen, and giving her an opportunity of judging for herself? Otherwise they knew what the result would be. She would die a painful death. The arm would swell, and the poor woman would be miserable for the rest of her life. He certainly thought, from what he had heard to-night, that he, as a practical surgeon, was justified in recommending the operation which Dr Beatson had recommended for cases inoperable by other means, and by which patients had been saved. He intended to try it when occasion arose; if this woman wished it he intended to try it on her. With regard to the second case, they had only to look at the cast taken before treatment and the cast recently taken. No one could say there was a cure; but there was such an improvement that he thought it could very well be put side by side with the first case.

*Mr Joseph Bell* said that since he saw so many inoperable cases at the Longmore Hospital, this paper had given him the deepest possible interest. The first case he thought one of the most remarkable cases he had seen in his life. He knew Dr Beatson well. His diagnosis of cancer was thoroughly good. Other people had seen it. And yet that woman was cured. He did not say it would not come back. He quite agreed with *Prof. Chiene* that such a case ought to have a chance either of thyroid treatment or excision of the ovaries, or both. Of course,



they all knew that the younger the patient the more inoperable the cancer was likely to be. The worst case he ever saw was that of a girl of 23. There was one operation. She recovered from it, but had a recurrence, and died in twelve weeks. She was a patient of Mr Syme's. He (Mr Bell) had her breast in his collection. The cases that came under his notice were generally inoperable and in old people, and his friend Dr Beatson would excuse him if he said that he had seen a good many very like No. 2, in which in the gradual process of shrinkage of the whole woman the glands diminished and the tumour diminished without any special treatment. No. 3 was very remarkable, but he had seen one or two even more remarkable. He watched one old lady in private for nearly fifteen years. She ulcerated, but did not lose flesh much, and not a gland was affected. There was no doubt of its being cancer. She died eventually of bronchitis. He was not so much impressed, therefore, with Nos. 2 or 3. But No. 1 was a remarkable bit of information, and he would like to add his own little quota of thanks for the admirable manner in which Dr Beatson could teach. He was able to follow every word, and that was not the case with all the papers one heard in that room.

*Dr James Ritchie* said he did not think Dr Beatson ought to be discouraged from operating on a case that had passed the climacteric. For some months past he (Dr Ritchie) had been studying a disease in which the ovaries seemed to have a trophic effect on bones and muscles, viz., osteomalacia. Fehling believed that the disease was a reflex trophoneurosis, having its source in the ovaries. Dr Ritchie did not believe that this expressed the whole cause, but the ovaries played a very important part. Many cases were cured by removal of these organs. Several cases had occurred after the climacteric in which the disease had made continual progress, but was cured by removal of the ovaries; one was cured about the age of 70. They would watch with very great interest the development of Dr Beatson's work. Dr Beatson had given them further proof that the generative organs had a profound influence on nutritional changes in the system generally. He did not think that they were warranted in going further than that.

*Mr Harold J. Stiles* said he need not say with what extreme interest he had listened to Dr Beatson's remarks on the treatment of cancer. He should like to congratulate him on the very clear way in which he had put his case, and also the very modest and cautious way. If he had betrayed any incaution, he (Mr Stiles) thought it was only in the field of hypothesis. One could not dispute the facts after seeing the first case. Dr Beatson was kind enough to show him the case about Christmas time, and even at that time he failed to discover any cancer. He did not believe that at present there was any cancer there. There were one or two little nodules, but these he had often seen. A little keloid

tumour often developed at the seat of the wound. It was only the other day that Mr Chiene removed a carcinoma of the mamma, one or two nodules in which were undoubtedly carcinomatous, but others consisted of thickened cicatrix. He examined all the nodules with much interest. Some were undoubtedly cancer, but others were doubtful. He did not mean that the first case was not a case of cancer, but only to show that the little nodules were not cancer. He had not an opportunity of seeing the specimen removed at the time of removal of the ovaries until to-night, and from a cursory examination he had no hesitation in saying that it was a case of carcinoma. To come to Dr Beatson's theory, he (Mr Stiles) agreed with him in not regarding the so-called cancer organism as a parasite. During the last six months he had had some cases illustrating this in a very remarkable degree. They were not to be found in every case, but if one examined a large number of tumours, now and then they got hold of a specimen which showed them exceedingly well. He had photos. which he hoped to show either to this or to some other Society, with the hope, he would not say of proving, but of substantiating, his disbelief in these so-called cancer bodies. If, then, they went back to the foundation and asked what the cause was, he was afraid he did not feel inclined to attribute its outbreak to any influence on the part of the generative organs. He believed the disease broke out independently altogether of the genital organs. He was not surprised to hear Prof. Simpson say that cancer was very common in women whose ovaries had been removed. If the healthy organs had an influence like this, why should the disease in the mamma be so localized? Why should it not break out over the whole mammary area? He did not think Dr Beatson should use the word ova. It was simply that the cells referred to were more eubryonic in type, and went on proliferating. Each tumour had more or less its own kind of malignant cell. All malignant cells were not exactly the same, but varied much in different cases. There was a birth-rate and death-rate of cells. They went on together. In the more slowly growing, in the atrophic tumours, and more especially in the ulcerating tumours, the death-rate was almost as rapid as the birth-rate, so that the tumour growth was very slow. He thought the influence of the removal of the ovary was simply to turn the scale between birth-rate and death-rate. It might check the former, and at the same time encourage degenerative changes or death-rate. It was much more likely to do that in the case of the mamma. He considered the first case a cure; but he believed the reason was that she was a young woman and had recently borne children. They knew that the ovaries had a decided influence on the functional mamma. He did not think they could expect that removal of the ovaries or testicles would have any curative effect on malignant tumours growing from other than the generative organs. He thought the treatment was well

worthy of trial, more especially in younger women, and he thought also it should be tried on elderly women. It might diminish the activity of the proliferation and increase the death-rate, and so prolong the life of the patient.

*Dr R. A. Lundie* said that, in common with all who had spoken, he had listened with extreme interest to Dr Beatson's paper. He thought they were indebted to him for taking them so fully into his confidence, for giving them the whole history of his thought and work on this subject. He felt that that gave to his paper very much greater vividness and interest than it would otherwise have possessed to him (Dr Lundie). The position in which they stood with regard to all cases of cancer, except those which could be completely removed with the knife, was a very distressing one. They had to say to the patient that, according to the current view, cancer was incurable. They took away all glimmer of hope. He had noticed in recent years occasional cases in medical journals, apparently undoubtedly cancer, that had recovered. He hailed these cases with pleasure, as enabling him to say that such cases occasionally did recover. They had all seen to-night one case of undoubted cancer that had recovered, and he thought that would help them to try not to take away all hope from cancerous patients. In the treatment of cancer and all other diseases the medicine of most potency of all was, in his opinion, the medicine of hope; and Dr Beatson attacked these cases of his, of which they had seen the results to-night, not only with new treatment, but above all, with the feeling of hope in the success of his attempt. He (Dr Lundie) did not think they were at all in a position to say yet whether removal of the ovaries or the administration of thyroid had a decided influence on the cases. He thought it quite conceivable, at least, that the hope which from his hope Dr Beatson had succeeded in infusing into his patients had, at all events, a most important influence on the result; and he thought they were especially indebted to him for showing that there were cases of cancer which did recover, so that they might try all in their power, by his or other methods, to induce hope in their patients.

*Dr Norman Walker* said he would make just a few brief remarks. One was to express satisfaction that it was only in inoperable cancers that Dr Beatson recommended this treatment. One saw so many cases in which other treatment had been tried before the surgeon had his opportunity that it was very desirable to point out clearly that this method should be tried only in cases beyond reach of the surgeon's knife. The second point was in connexion with the third case shown to-night. That case seemed to him to be one of rodent ulcer; as did also Mr Bell's, and not ordinary cancer. (Mr Bell—"Mine wasn't.") Mr Bell's case had, at any rate, the clinical history of rodent ulcer. Dr Beatson's had the features of it, both clinically and microscopically. He thought

that in that case removal might have been quite well done with the knife.

*Dr Beatson*, in reply, said he thanked them very much for the very kind reception they had given to his paper. They had entered into the spirit in which he hoped it was written. He need not go into detail, because the different speakers had gone over much the same ground. First of all, as to Prof. Simpson's reference to Spencer Wells' case of carcinoma following removal of both ovaries. He thought if these cases were looked into they would find that the carcinoma had appeared within a few weeks of the operation, at any rate in several cases. That afforded ground for saying that in all probability the disease was there at the time the operation was done. Cancer did grow rapidly, but not, like a mushroom, in a single night. He had looked into these cases. One died five years after operation, but they were not told how long the cancer had existed before she died, which was a very important point. The disease ran a slow course in some, in others a more rapid one. He thought that in the majority of those cases of Spencer Wells' it had been early and rapid, and also in the abdomen. He thought there were one or two in which mention was made of secondary deposits in bone and other organs, but it was not said whether they were of the same kind as those in the abdomen. He quite admitted with Prof. Simpson that his view about carcinoma was problematical and theoretical. But it was a point which might be worked out. At the present day much was being done on the subject of development. An ovary that was to all appearance healthy might have changes, especially chemical, going on that were not apparent. They must have a standard to which the ovary, when examined microscopically and chemically, must come up before they could say it was distinctly healthy. In regard to Mr Bell's remarks on the second case, in which he said that shrinkage of the tumour accompanied shrinkage of the patient generally, he watched that point. The patient on admission weighed 7 st. 8 lbs. Her weight at present was 7 st. 7 lbs., so that practically it had been a local shrinkage. Whether the shrinkage was entirely due to the atrophy of glandular substance alone he could not say. Each mamma was undoubtedly atrophied, but the general diminution in the size of the tumour, as apparent from the two casts, was very striking. He quite agreed with Prof. Chiene that the practical point was the one they had to look to. At the same time, he would not have it go forth that he stood there to advocate removal of the tubes and ovaries or testicles for cancer. It was a dangerous thing. But he hoped that cases on which it might be tried would be carefully noted, and the results carefully published. He had done the operation in some cases of carcinoma of the uterus. It took months before these cases showed appreciable change. They were all cases in which local removal was out of the question. To one he was giving pure

ovarian extract. She was brought in very much exhausted from hæmorrhage last October. Since removal of the ovaries there had been a very remarkable condition of the tissues. The hæmorrhage had ceased, and the tissues had assumed a dried-up, almost shrivelled appearance, with freedom from discharge. Her weight was going down, and she had undoubtedly that yellow cachexia that they saw in those cases. Yet she had been keeping remarkably free from offensive fœtor and sacral pain. Altogether he was watching the case with great interest. He had not felt he had sufficient grounds in the case third shown for doing anything local. There were cases in which cutting into the tissues did undoubtedly favour the spread of the disease. If there were other cases he would, if the Society were willing, be very pleased to bring them before it.

### Meeting X.—June 3, 1896.

Dr ARGYLL ROBERTSON, *President, in the Chair.*

#### I. EXHIBITION OF PATIENT.

*Mr C. W. Cathcart* showed a patient who, he said, illustrated a point which was unknown to him until he had seen one or two examples of it this winter. In certain cases of tertiary syphilis the sore presented characters which one was always accustomed to consider tubercular—viz., marked undermining of edges of sore and irregularity of the scar. Of the other two he had seen, one was on the arm and the other on the neck. If one had not really been on the look-out for it in the ward, one might have taken it for tuberculosis. They healed up without any trouble under antisypilitic remedies. The patient had only been in the ward two days, and there was a great difference in the sore. The history was that three years ago he had syphilis; and now he had got, besides this curious undermining of the skin, a gumma forming on the clavicle, and another on the scalp.

#### II. DEMONSTRATION OF INSTRUMENTS.

1. *Dr Logan Turner* explained and demonstrated DR KIRSTEIN'S METHOD OF AUTOSCOPY OF THE LARYNX AND TRACHEA, OR DIRECT EXAMINATION WITHOUT THE AID OF THE LARYNGEAL MIRROR, and read the following notes:—

The principle of this method of examination consists in bringing the interior of the larynx and trachea *directly* under the eye of the observer without the use of the laryngeal mirror, and consequently

without dealing with any reflected image. The method is called autoscropy; the instrument employed is the autoscope.

Five conditions appear to be necessary for the satisfactory carrying out of this examination:—

1st. There must be a straight line between the aperture of the mouth and the interior of the larynx and trachea: the angle which naturally exists in the ordinary posture of the head, between the buccal cavity and the assumed upward prolongation of the trachea, must be got rid of. This is done by raising the patient's head until it forms an angle of about  $60^\circ$  with the axis of the trunk.

2nd. All prominences which project into the lumen of the canal and obstruct the direct view downwards must be effaced: the base of the tongue and the epiglottis are the two structures which thus interfere with the line of vision. The base of the tongue is kept in the required position by a spatula, which must press upon the posterior part of the tongue immediately *in front of* the epiglottis. The epiglottis is raised and drawn forwards by this same pressure on the base of the tongue, and by drawing on the glosso-epiglottic ligament which so closely unites the epiglottis with the tongue. In operating on the larynx by this method a spatula is introduced *behind* the epiglottis, and in this way it is removed from the line of vision.

3rd. The larynx and trachea must be efficiently illuminated: this is most frequently done by an electric lamp placed in the handle of the autoscope, though it may be obtained instead by an electric lamp attached to a forehead band.

4th. The patient must be able to bear the necessary pressure on the tongue.

5th. The examiner must have some experience and dexterity in the manipulation of the instrument.

These two last conditions must be considered to some extent together, for while many patients are quite unable to submit to the amount of pressure required for depressing the tongue and drawing forward the epiglottis, much discomfort may be superadded by want of dexterity on the part of the examiner.

*The Instrument.*—*The spatula*, of nickel-plated palladium, which can be cleaned by boiling, is 14 cms. in length, slightly hollowed out on its upper surface, and slightly curved backwards at its distal end, where it is notched to receive the central fold of the glosso-epiglottic ligament; it is fixed at right angles to the handle of the autoscope. It is provided with a small sliding plate, which is adjusted to the proximal end of the spatula, the two together forming a short tunnel 4-5 cms. in length, through which the light is transmitted, and through which instruments

for operative purposes may be passed. The spatula for introduction behind the epiglottis is longer, and is straight in its entire length.

*The handle* is provided at the end which receives the spatula with an electric lamp, the rays from which, by means of lenses, are directed along the spatula into the cavity of the larynx; the wires from an electric battery are attached to the other end of the handle, and by means of a button the current may be made or broken at will.

*The Examination.*—The examiner stands in front of the patient, who is seated on a chair with the head in the position already indicated. All false teeth should be previously removed; the spatula, slightly warmed, is then introduced with the electric current in contact, so that the buccal cavity is illuminated, thus permitting of an accurate placing of the end of the spatula immediately in front of the epiglottis; the handle is raised until the proximal end of the spatula, covered with the sliding plate already described, comes in contact with the teeth of the upper jaw; simultaneously its distal end presses the base of the tongue downwards and forwards. In a satisfactory case the examiner, looking along the spatula, will observe the epiglottis being raised and drawn forwards; then the arytenoid cartilages will come into view, the posterior wall of the larynx, the false and true cords, and, lastly, the trachea, and even its bifurcation may be visible.

No violence must be used; the movements employed for depressing the tongue must be rapid and certain, but not sharply or painfully carried out, and considerable experience is necessary for satisfactorily conducting the examination. In difficult cases, after preliminary failure, the mucous membrane may be sprayed with cocaine, and where the spatula is introduced behind the epiglottis and an operation contemplated, then cocaine should be employed.

There appears to be considerable variation in the amount of success attending this procedure. While many patients are quite unable to bear the pressure necessary for the drawing forward of the epiglottis, others again bear it without any feeling of discomfort. With the former it is impossible to conduct the examination satisfactorily; with the latter a good view of the interior of the larynx may be obtained; but even with these this is by no means always the case, because careful and repeated manipulation fails to draw the epiglottis sufficiently forwards even to permit of a view of the arytenoid cartilages. Dr Kirstein, as quoted by P. Bruns, saw the whole of the larynx in only one-fourth of the cases examined, the posterior part of the larynx in one-fourth to one-half of the cases. In three cases of laryngeal simple tumours recorded by Bruns himself, it was possible in one case to

remove the greatest part of the tumour by the direct method; in the second case only the posterior part of the tumour could be seen; and in the third the growth could not be seen at all, so that in these two last cases the growths had to be removed in the usual way with the use of the laryngeal mirror.

Autoscopy will not take the place of ordinary laryngoscopy, nor does Dr Kirstein suggest that it will; but he looks upon it as a valuable and real aid in diagnosis and treatment. In the case of children, who so frequently resent the ordinary method of examination, the experience of Bruns has been that they can be much more satisfactorily examined by the direct method. Kirstein in operating upon children by his method administers chloroform. The head is then drawn over the edge of the table and is held there by an assistant. The autoscope is held in the operator's left hand, the child's head being gradually raised or depressed until the required position is obtained, and then the necessary operation is carried out. The same position is employed by him for the mere examination of infants and young children, but without the aid of chloroform.

The instruments used for operating with the autoscope resemble in form the instruments used for the nose, the handle being fixed at an angle with the stem. For the larynx they measure from the bend to the point 20 cms. in length, while those for the trachea are somewhat longer. In other respects they do not differ from the instruments in ordinary use.

One of the earliest descriptions of this method of laryngeal examination will be found in the *Berl. klin. Wochenschrift*, 1895, No. 22; while the most recent paper by Dr Kirstein on this subject is in the *Annales des Maladies de l'Oreille*, etc., for March 1896.

2. *Dr Argyll Robertson*, the President, showed INDIAN INSTRUMENTS FOR COUCHING CATARACT. He said he was indebted to the kindness of his friend Surgeon-Major Jennings for the opportunity of exhibiting to them three specimens of the instrument employed by native oculists in India for the operation of couching cataracts. Dr Jennings had informed him that he had considerable difficulty in getting specimens of the instrument, as the quacks there were unwilling to give them up. A man, however, lately gave him some specimens, of which he enclosed three. They were of copper, and often very dirty. The quacks generally made an incision with a bleeding lancet and shoved the lens out of place with one of the enclosed weapons, withdrawing it when they had reached the neck or shoulder. The portion of the instrument introduced into the interior of the eye, and by means of which the lens was depressed, was shaped very much like the blade of a bayonet.



## III. ORIGINAL COMMUNICATIONS.

## 1. THE RÔLE OF THE CARBOHYDRATES IN DIETETICS.

By W. G. AITCHISON ROBERTSON, M.D., D.Sc., F.R.C.P. Ed., F.R.S.E.

## GENERAL CONSIDERATIONS REGARDING DIET.

*Diet Tables.*

## 1. Full or ordinary diet, Royal Infirmary, Edinburgh.

Meat, . . . . .	8 ounces (= 1½ oz. water-free albumin).
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Vegetables, . . . . .	8 "
Bread, . . . . .	16 "
Barley, rice, or peas, . . . . .	1½ ounce.
Sugar, . . . . .	1¼ "
Salt, . . . . .	¾ "
Butter, . . . . .	1 "
Porridge and milk if desired.	

## 2. Public diet (Moleschott).

Proteids, . . . . .	130 grams.
Fats, . . . . .	84 "
Carbohydrates, . . . . .	404 "
Salts, . . . . .	30 "
Water, . . . . .	2800 "

## 3. Diet of European armies.—Water-free food in ounces.

	English.	French.	Russian.	Austrian.
Albuminates, . . . . .	3·86	4·33	4·02	3·73
Fats, . . . . .	1·30	1·27	1·09	1·64
Carbohydrates, . . . . .	17·43	18·04	19·62	17·0
Salts, . . . . .	0·81	1·0	1·50	1·0

## 4. Sailor in British Navy.

Salt meat, . . . . .	9 ounces.
or	
Fresh meat, . . . . .	4½ "
Carbohydrates, . . . . .	26½ "

## 5. Prisoners at hard work, and confined for more than three months;

36 ounces of food daily. Butcher's meat forms only a small proportion of this, amounting to 16 ounces per week.

6. The diet of the Trappist monks is vegetarian, and consists daily of—

Bread, . . . . .	17½ ounces.	} =	Proteids, . . . . .	2.186 ounces.
Beer, . . . . .	17½ „		Fats . . . . .	0.35 „
Vegetable soup, 2 plates.			Carbohydrates, 15.08	„
Green vegetables, 1 „				

From these tables it is manifest that the chief element in all dietaries is the carbohydrate one, the proteids forming but a small proportion, the fats and salts holding a still lower place.

While proteids are of first importance in building up the active tissues, the carbohydrates form, through their combustion, the energy or heat-producing agencies, or are stored up as reserves of such.

Proteids are notoriously less easily digested than carbohydrates, and it is affirmed that vegetable proteids are even less digestible than animal proteids. It is a matter of common observation that, unless we take active exercise when living on a diet rich in animal food, we soon get out of sorts, and become bilious or dyspeptic. In all likelihood this is due to accumulation of many extractives in the body. Where do the most of the proteids come from which we consume in our food?

Looking at the percentage composition of flesh, we see how little proteid material it contains, and one can easily calculate the small quantity of nitrogenous matter which is obtained in consuming the ordinary amount of meat.

*Composition of Flesh.*

	Ox.	Fowl.
Water, . . . . .	77.50	77.30
Solids, . . . . .	22.50	22.7
Soluble albumin, . . . . .	2.20	3.0
Insoluble albumin, . . . . .	17.50	16.5
Fat, etc., . . . . .	2.30	3.2

It is obvious, therefore, that in ordinary diets the nitrogenous matters are chiefly of vegetable origin, and so are usually consumed along with the carbohydrates in the form of bread, farinaceous materials, or vegetables.

While a mixed diet in which animal food is present may be, and probably is, useful, we see that it is by no means absolutely necessary. The proteids contained in farinaceous foods and vegetables are amply sufficient in most cases to replace the animal proteids, and I believe that when the organism becomes accustomed to the vegetable proteid there is little greater difficulty in digesting it than there is with the animal proteid. There could hardly be found stronger men than Scotch ploughmen of a century ago, and

their staple food consisted of porridge and milk. The prisoner's diet of to-day would be much improved did it contain more of the quondam ploughman's food, and many would leave prison in better health than is the case at present. Native Indians eat scarcely anything but rice, to which a little butter (usually rancid) is added, and rice is almost the poorest in proteids of any farinaceous food which we have (7.40 per cent.).

It is usually stated that when a food stuff so rich in one element is used alone very large quantities of it must be consumed, so as to get a sufficiency of that element in which it is poorest. Theoretically this is true, but in practice it is seldom observed. The Chinese or Indian eats by no means an extravagant quantity of rice; in fact, we should be inclined to call it very moderate. Then we have always the extreme and historical examples (like Alexis St Martin in questions of digestion) of Cornaro, who lived healthily and well for fifty-eight years, or till he attained the age of 103, on 12 ounces of food, chiefly vegetable, and 14 ounces of light wine daily; or of the still more abstemious Thomas Wood, a miller of Belaricoy, who lived happily for nearly twenty years on a daily pudding made of a pound of flour with water, no other fluid being taken.

I have no intention of posing as an advocate of vegetarianism, though I think there is a very great deal to be said in its favour. Of course, we know well the oft-alleged assertion that, owing to the structure of the digestive tract in man, he is capable of digesting both animal and vegetable foods, and therefore ought to make use of both. The capacity for digesting both of these varieties implies, however, that each can be perfectly used up in the human economy. We know of many hardy races who are almost, if not entirely vegetable eaters. Dr Parkes remarks that the meat eater and the man who lives on corn, peas, or rice are equally well nourished, and that the well-fed vegetable eater shows, when in training, no inferiority to the meat eater. Then, amongst animals, the largest and most powerful, either domestic or wild, are purely vegetable eaters,—as the horse, ox, elephant.

I think these facts are sufficient to show that perfect health may easily be enjoyed while animal food is excluded from the dietary; and at the same time they may serve to direct attention to one of the most important elements in a proper diet, viz., the *carbohydrates*. We have seen that they form the largest part in any scheme of dieting, and we remember that they form the staple food of young children and invalids. They thus merit, in my opinion, a greater regard than as yet they seem to have received from the clinician or physiologist.

Digestive troubles form the most potent factors in swelling the mortality tables of children dying under one year. This fact is more strikingly brought home to us when we recall that the

mortality of children is greatest from the fourth to the sixth month, and this period corresponds to the time when "hand-feeding" is usually begun, with its dire results in too many cases.

Again, in towns this practice of artificial feeding of children is far more general than in country districts, and this is strikingly shown in the Registrar-General's Returns. For instance, the death-rate per 1000 from diarrhœa in 1887 was, in rural districts 0.50; while in London it amounted to 0.90; and in the twenty-eight "great towns" together it formed 0.97. In 10,000 deaths amongst children in the city of Berlin it was found that 7646 had been artificially fed.

Apart from the question of infant mortality as the result of mismanaged feeding, there is the immense importance of this branch of dietetics in the many forms of indigestion and diseases of the alimentary organs in adults.

Statistics of hospitals and dispensaries prove that affections of the alimentary tract form by far the largest proportion of all diseases occurring during adult life, and these are only too frequently induced by unsuitability of food or irregularity in feeding.

In this paper I shall only deal with one branch of the carbohydrate group, viz., the digestion of starches (I have already in other papers treated of the digestion of sugars), though incidentally we may have to speak of sugars also.

#### *Résumé* OF THE CHEMISTRY OF STARCH.

The chemical constitution of starch may be represented in its simplest form by  $C_6H_{10}O_5$ . More probably, however, it is a multiple of this, and  $n$  ( $C_{12}H_{20}O_{10}$ ) would represent it more accurately,  $n$  being unknown, though probably never less than five or six.

When starch is heated by itself, or, as mucilage, if boiled with dilute acids, or when acted on by diastase, it becomes changed into an isomeric body—dextrin.

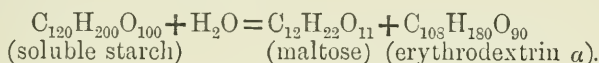
There are many varieties of dextrin. That which is formed earliest is termed soluble starch or amylo-dextrin. As dextrination proceeds erythro-dextrin is formed, and still later the achroo-dextrins. If ebullition with acids be continued, or if diastase be allowed to act for a long enough time on the dextrin, it becomes hydrated and changed into maltose, which in its turn becomes converted into glucose ( $C_6H_{12}O_6$ ) through the continued action of diastase or of dilute acids.

When a solution of iodine is added to starch, a deep blue colour is produced. Soluble starch in solution gives a violet colour with iodine. If dry, however, the colour produced is yellow, violet, or brown.

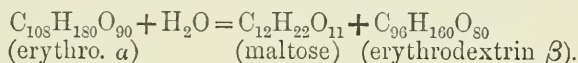
As the starch undergoes conversion this violet becomes a purplish-red and then a red colour with iodine, showing that the stage of erythro-dextrin formation has been reached.

Still later this red colour becomes lighter, till at length no coloration results from adding iodine solution. This is the stage of the achroodextrins. Of these there are many varieties differing from each other in their rotatory and reducing powers. The first to be formed,—achroodextrin  $\alpha$ ,—can still by the action of diastase be changed into maltose and glucose, as happens with amylo-dextrin and erythro-dextrin.

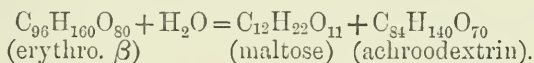
The formation of dextrins has been explained thus. The constitution of soluble starch is probably represented by the formula  $C_{120}H_{200}O_{100}$ . Under the influence of the diastase of malt it assimilates a molecule of water, and so forms a molecule of maltose, the rest going to form erythro-dextrin  $\alpha$ .



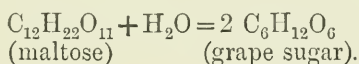
On further hydration another molecule of maltose is formed, and erythro-dextrin  $\beta$ , which has a less molecular weight.



By a similar process achroodextrin results—



There is a difference of opinion between chemists as regards the further changes which occur. The most recent and careful investigations seem to leave no doubt however but that the hydration process goes on till only maltose is left. Each of its molecules in turn takes up a molecule of water through the continued action of diastase or dilute acids, and splits up into two molecules of grape sugar—



### *Digestion of Starch.*

Ptyalin is the ferment in the salivary secretion which converts the starch granules into achroodextrin, maltose and glucose. Through the continued action of this ferment maltose is slowly split up into glucose. The amylolytic action of saliva is destroyed by high temperatures, as by boiling; while at low temperatures conversion becomes slow, and ceases at  $0^\circ C$ . At the normal blood heat ( $38^\circ C$ .) starch is converted by ptyalin very rapidly in an alkaline solution. It is said that small quantities of hydrochloric acid suspend and rapidly kill the ferment. On the other hand,

it is affirmed that its action goes on in neutral as well as in slightly acid solutions. In strongly acid or alkaline solutions its action soon ceases.

The question as to the activity of the salivary ferment in acid solutions is of great importance as regards the digestion of starch in the stomach. If hydrochloric acid be poured out early in digestion, the alkalinity of the swallowed saliva will be soon neutralized, and the activity of the ptyalin must soon come to an end. It is almost certain, however, that the hydrochloric acid in the stomach combines at once with the proteids during the earlier stages of digestion, and so, no free acid remaining, saliva still continues its conversive action on starch. This condition lasts for a time varying from three-quarters to two hours. At the end of this period the proteids have combined with as much acid as they require, and now free hydrochloric acid appears in the gastric secretion. The conversion of starch in the stomach then ceases, as ptyalin is destroyed by the acidity of the gastric juice in full digestion. There are thus two periods in gastric digestion—(1), when saliva still acts; and (2), when pepsin is alone active.

What degree of acidity hinders or kills the ferment ptyalin? Do the various acids differ in their power of destroying ptyalin?

These questions seem to me of great importance. During the earlier stages of digestion, though no free hydrochloric acid is present, the gastric secretion is often acid from the presence of lactic, acetic, malic, or tartaric acids, or from acid salts, taken along with the food.

Does ptyalin still act during this period? If it only acts in alkaline or neutral solutions, then its action will be limited to the time during which food is being masticated and swallowed; or if the food be itself acid, its converting action ought to be entirely inhibited. In the stomach its action will only go on till the alkalinity of the saliva is fully neutralized.

In nearly all cases, therefore, ptyalin must be entirely destroyed before the contents of the stomach escape into the duodenum. Only in these cases where the acid has been so deficient that conversion of starch has gone on uninterruptedly in the stomach, can ptyalin escape destruction. Nor, indeed, is it necessary that ptyalin should be preserved in normal conditions, for soon after the contents of the stomach escape into the duodenum their acidity becomes neutralized, and they then encounter the pancreatic secretion, which is very much more powerful than the saliva in its diastatic action on starch, as it transforms it almost immediately into maltose and glucose. This converting ferment of the pancreas, amylopsin, can act on raw starch at the temperature of the body, giving rise to the production of several dextrins which have a reducing power on alkaline solutions of copper as well as maltose and glucose. The pancreatic secretion further changes maltose and dextrins into glucose. This hydration

process is slow, however, and is much assisted by the action of the *succus entericus*.

The *succus entericus* has only a very slight action on starch (it is even denied that it possesses any), and it takes many hours' digestion with it even to produce a small amount of erythro-dextrin and soluble starch. The small intestine possesses, however, a powerful hydrolytic action on maltose, much more so than on soluble starch, dextrans, or even cane sugar. This property is chiefly resident in the agminated and solitary glands of the small intestine. The physiological actions of the pancreas and *succus entericus* are consequently mutually dependent. Starch under pancreatic proteolysis becomes changed for the most part into maltose and glucose. The former then encounters the intestinal secretion and undergoes hydration and splitting up to form glucose. In this way the whole of the starch is at length changed into glucose.

A starch-converting ferment is, however, widely diffused throughout the tissues of the body, and Magendie long ago showed that blood serum possesses this amylolytic power. It has also been recently shown that if solutions of starch be slowly injected into the tissues they are soon converted and entirely absorbed.

#### *Digestion of Starch during Infancy.*

Are the digestive processes of the young child materially different from those found in the adult? Can infants fully digest and absorb amylaceous matters?

This question has been carefully investigated, and it has been found that though starch can in very small amount be digested from the earliest period of life, yet it is a slow process. For the first two months of the child's life the amount of saliva secreted is very small, and we have all noticed how dry the mouth is in healthy infants. During the first month of the child's life the total secretion of mixed saliva is only about 1 c. c. in twenty minutes. The secretion rapidly increases after the first month: till at the third month the rate is about 1 c. c. in two minutes. Along with this scantiness in secretion of saliva, its proteolytic action is slow, and in a child seven days old, starch solution only showed a commencing conversion after it had been acted on for four minutes by the saliva. Because of this scanty secretion and slow action, many have thought that the salivary secretion in infants possessed no diastatic action on starch.

Ptyalin does not appear in the saliva to any marked extent till the sixth month; that is to say, till the eruption of teeth begins. At this time also saliva is secreted in great abundance, though there is a larger admixture of mucus, probably from the irritation, than at other times.

It is not, however, till nearly at the end of the child's first year

that the amylolytic action of the saliva reaches its full amount and power. There is no essential difference between the gastric digestion of infants and adults.

The pancreatic secretion possesses no converse action on starch during the first month of life. During the second month this power is developed, and is well marked at the third month. But, as with the salivary glands, so here: the full development of the diastatic action is not reached until the end of the child's first year.

At an early period of life, moreover, the number of glands in the intestinal canal is relatively small. As the child grows older, so does the number of glands increase. The development of the glandular system advances *pari passu* while the lymph vascular system decreases in importance.

## THE CONSTITUTION AND USE OF INFANTS' FOODS.

### INFANTS' OR INVALIDS' FOOD.

WE come now to consider foods designed for the special use of infants or invalids.

#### *Condensed Milk.*

This is by no means a starchy food, yet I may be pardoned for mentioning it, as it is undoubtedly by far the most universally used form of infants' food. It is easy to see why this is so, for owing to its small bulk, and consequent convenience in carriage, the length of time which it keeps, and the ease with which it can be made ready, its use is made general.

It is certainly for many children a good substitute for mother's or even for fresh cow's milk, and specially so for children during the first month or two after birth, with whom it nearly always seems to do well. But still this does not warrant us in recommending its general use, for many children assimilate with impunity food which is most deleterious to infants as a class.

This milk is prepared in two forms—

- (1.) Simply condensed and unsweetened.
- (2.) Condensed and with cane sugar added to preserve it.

The first consists in merely evaporating ordinary cow's milk down to one-third or one-fourth its original volume. In only too many cases, however, the milk has previously undergone a partial, or even an entire, skimming to remove the cream before it has been concentrated.

This forms a thick, syrupy, pale-yellow fluid, and merely requires the addition of water to restore it to its "original condition" (?) it is said. This preparation is not so largely used as the second, as, on account of there being no preservative added, it does not keep sweet for more than two or three days after the tin is opened. If kept longer than this, or in warm weather



when organic fluids decompose rapidly, there are all the risks of giving the infant a fluid which is beginning to undergo fermentative changes, and we know how common a cause of infantile diarrhoea tainted cow's milk is during summer or autumn.

The sweetened variety is the one so universally used. It is a generally accepted fact that many infants fed on this variety rapidly put on fat. Many of these children are flabby and soon show signs of rickets. Gastric and intestinal catarrhs frequently occur in them, and it is rare to find that they have uninterrupted good health. My own experience agrees with the usual opinion that such feeding makes children less able to withstand the usual diseases of infancy, or renders the convalescence from them slow and unsatisfactory.

Of the large number of rickety children found in all large towns, a very great proportion occurs amongst those who have been brought up on artificial food, such as condensed milk. In rapidly growing children the need of nitrogen must be satisfied in order to obtain proper development; but while in cow's milk the proportion of nitrogen to carbon is as 1 to 12, in preserved milk it is 1 to 20. The large amount of carbon given in such food explains why there is such a rapid deposition of fat, but the needs of bone and muscle must remain unsatisfied by such a food.

I examined several specimens of condensed and sweetened milks as they were found in the market, and subjoin the figures found in a few.

1. This was found to contain 13·13 per cent. of milk sugar. I then estimated the amount of cane sugar present by the method described by Pavy, and found it present to 41·6 per cent.

2. In another specimen of condensed milk I found the reaction to be acid, and that it contained 11·7 per cent. lactose and 43 per cent. cane sugar.

3. Another was said to be partially peptonized and condensed. It contained 12·63 per cent. lactose, and 33·84 per cent. of a substance having a reducing action on Fehling's solution and which was probably maltose. It contained, likewise, dextrins and fat, but no unchanged starch.

The first two of these preparations contain, therefore, a very large amount—nearly one-half of the total constituents—of a substance so difficult of absorption as cane sugar. This improper food is also increased in amount as the child grows older, not only by lessening the dilution of the milk but by giving him more of it, and thus the risks of this sugary food increase *pari passu* with the age of the infant.

In spite, therefore, of the general use of such sweetened condensed milks, I have no hesitation in stating that their use constitutes a form of injudicious feeding. The proportions of the food constituents are so altered that it has little resemblance to ordinary breast milk. Simple condensed and unsweetened

milk is not in itself to be condemned, though certainly far inferior to fresh cow's milk, for the relative proportion of the constituents remain pretty much as they were originally on again diluting the milk. The constant use of the variety sweetened with cane sugar is, however, to be strongly deprecated.

It is a common practice amongst the poor to add starchy materials—as corn-flour, arrow-root, pounded biscuit, bread, etc.—to the milk which is given to the child. This is done with the idea of thickening the milk and making it thus more nutritious, for it is a popular idea that cow's milk is in itself not a sufficient food. They forget, meanwhile, that if the child were fed from the breast it would receive no other added food.

The physiological processes in the infant show that starchy matters must be very imperfectly digested by it, owing to the extreme feebleness and the scantiness of secretion of the diastatic ferments at an early period of life. All authorities agree in stating that farinaceous materials are quite unadapted and hurtful to the young child.

The only carbohydrate which a naturally-fed child receives is sugar of milk. There is no substance in milk which in the least corresponds to starch. It is only at the sixth month that the diastatic ferments of the saliva and pancreas are secreted to any extent, and certainly no pure starchy food ought to be given before this age. The full action of the saliva and pancreatic secretions on starch is not attained till the end of the first year, and it is only then that we may allow ordinary farinaceous food, as rice, corn-flour, etc., to be given. If we permit the use of such food before this age, then assuredly the starch should be pre-digested. By this I mean that unchanged starch must be rigidly excluded from the dietary and only given in a readily assimilable form, as after partial or complete conversion into dextrins, maltose, and glucose. I have already shown that glucose and some dextrins are directly absorbed, while maltose is not so, but requires to be changed into glucose first. Starch should be wholly converted into dextrins and dextrose when added to milk for infants of from five to six months.

At an early age it is sometimes found to be advantageous to add a carbohydrate to the dietary when the child is not thriving on milk alone, or in those cases where cow's milk sets into too firm a curd even when diluted with water. This firm curd is not well acted on by the gastric juice, and is rejected by vomiting. When a farinaceous material is added, however, the density of the curd is lessened, and so its digestion is rendered easier. In such conditions only the easily assimilated carbohydrates should be used, and I would recommend dextrins and dextrose, which, being easily absorbed, afford small opportunity for fermentative changes to occur.

As the diastatic power of the saliva and pancreatic secretions

increases, we ought gradually to lessen the degree of starch conversion so as to stimulate more the secretion of these ferments. Thus, after the age of six months, I would only partially convert the starch, so giving a mixture of dextrins, maltose, and glucose. As the child grows older the degree of conversion ought to be still further gradually lessened, thus reducing the amount of glucose, while the quantities of dextrins (of lower variety) and maltose are increased relatively, till, at the end of the first year, we may allow almost natural well-cooked starchy foods to be given. To corroborate this, it has been shown by analysis that as lactation proceeds the amount of albumin in human milk diminishes, while the amount of carbohydrate increases, the amount of fat remaining the same. This goes to prove that as development proceeds the need for nitrogen becomes less, while the necessity for carbon increases.

This leads us, therefore, to the consideration of the so-called "infants' food." The food which is suitable for the young child is unsuitable for the same child at a later period, and so in order to be able to direct the dietary of the infant we must know of what these foods are composed.

Many different preparations are sold to the public under the name of "babies'" or "infants' food." Some, through their own merit, but more through judicious or persistent advertising, have become widely known and largely employed as additions to, or substitutes for, cow's milk.

The foods in themselves may be good enough, but may be unsuited to the age or condition of the children to whom they are given. The infant may thrive on a certain food for a time, but afterwards begins to fall off, even though it is taking the same food readily. If so, there must be some dietetic error, and we will probably find that it is in the character of the artificial food. This, though it nourished the infant well enough at an earlier period, does not form a sufficiently nourishing food for the same child when older.

Again, if we continue to give predigested food for too long a time, the secretions of the alimentary canal lose their distinctive digestive properties to a large extent through disuse of the glands. Thus on changing the food to one not previously digested, rapid emaciation follows, because the digestive secretory glands, having been out of action for some time, require an interval during which to regain their functions.

The constitution of some of these foods we know, but of many the nature of the ingredients is kept secret. In medicine anything which savours of quackery is eschewed. Why should it not be so likewise in dietetics, especially when they concern infancy? The whole after-life may be made or marred by the nature of the food partaken of during the active development of the child.

In all foods designed for the use of infants we must have the

four elementary principles—proteids, fats, carbohydrates, and mineral matters. These must be combined in certain relative proportions so that the child may get a sufficiency of each and yet not an excess of any one. Too small or too great a relative amount of any leads ultimately to ill-health. In the first case some of the tissues are starved, while in the second all cannot be absorbed, and so intestinal irritation and general ill-health result.

It is, however, to the carbohydrate constituent of these foods that I desire now to devote attention, as this forms by far their most important element, and, as I have pointed out, requires the greatest care in administration.

#### EXAMINATION OF INFANTS' FOODS.

In most of the analyses of these foods the results refer to them as they occur in commerce, and not to the food as it exists after being prepared for use. These two analyses are very different in many cases. My investigations were made in order to determine in what condition the starch was after the food had undergone the process of cooking.

In preparing these for examination I followed the directions given with each food, only instead of using milk as the menstruum (which is sometimes recommended), I used ordinary water.

I generally made a 5 per cent. solution of the food, but when this gave too thick a mucilage I reduced it to a 2 or even to a 1 per cent. solution. Having allowed it to cool, I then tested the mixture for the presence of unchanged or soluble starch, erythro-dextrin, achroodextrins, or sugar. I have named all those matters which have a reducing effect on Fehling's solution for brevity "glucose," but which may be glucose, levulose, maltose, lactose, or reducing dextrins. In each case I have calculated them as dextrose.

A definite amount of the solution was then heated on the water-bath for two hours at 140° F., five minims of a dilute solution (1 in 10) of sulphuric acid having previously been added to it. By this proceeding any cane sugar present in the food underwent inversion. This solution was again allowed to cool, any loss from evaporation made up, filtered, the condition of the starch again examined, and the amount of reducing substance again estimated.

In those cases where malt or malt extract was supposed to be present, instead of boiling (which would have destroyed the diastase) I simply warmed the solution, and having set it aside in a warm place for half an hour, examined the condition of the starch and estimated the "glucose" in it.

For obvious reasons I have refrained from mentioning the foods by name.

1. *A Milk Food* (N. M. F.).—This food is in the form of a

yellow powder, extremely difficult to mix with cold water, and when so giving a muddy solution, having a faintly acid reaction. It has a very sweet taste, resembling that of ground biscuit.

The directions bear that it should be mixed with cold water and then boiled for a few minutes with continuous stirring.

Having made according to the directions a 2 per cent. solution of—

(1.) Boiled food, I found that it contained unchanged starch, soluble starch, some erythrodextrin, and 4.5 per cent. of glucose.

(2.) Having acidified and heated the solution for two hours, it then contained much unchanged starch, erythrodextrin in large amount, and glucose to 38.5 per cent. Lest there should have been malt or malt extract in this food, and which the boiling would have destroyed, I prepared a solution but only heated it to 100° F.

(3.) Solution only heated, not boiled—unchanged starch, trace of erythrodextrin and glucose, 6.65 per cent.

(4.) This solution, kept hot for thirty minutes, gave similar results.

(5.) A cold extract of the milk shows the presence of unchanged starch, traces of erythrodextrin and glucose 4.35 per cent.

This food clearly contains a large amount of cane sugar (34 per cent.), which must, by the method of preparation, be administered as such. The acidity was too feeble and the heat insufficient to have converted entirely the starch, except perhaps into some early formed dextrins; nor did there appear to be, after boiling, any dextrins present which might have been converted more readily. We must conclude, therefore, that this food contains much unchanged starch and cane sugar.

2. *A Soluble Food for Infants* (C. F.).—This is in the form of a very fine light yellow powder, freely soluble in water, and having a neutral reaction. It is said that this food is partially predigested by pancreaticine.

*Directions.*—The food is to be dissolved in a definite amount of cold water; then it is to be gradually added to an equal amount of boiling water, stirred till it boils, and boiled for two minutes.

Having made a 2 per cent. solution of—

(1.) Boiled food, it contained unchanged starch only, and glucose 20 per cent.

(2.) Acidified and heated at 140° F. for two hours. Unchanged starch and soluble starch, erythrodextrin in large amount; glucose now 23 per cent.

To see if the pancreatic ferment were active, or if invertible sugar were present, I prepared a solution, but did not boil it.

(3.) Solution made with hot water (not boiled), and examined at once, showed only unchanged starch; glucose 18.5 per cent.

(4.) Same solution kept warm (100° F.) for thirty minutes, showed

unchanged starch, small amount of erythrodextrin, and glucose increased to 25·6 per cent.

(5.) A cold extract shows unchanged starch, erythrodextrin in traces, and glucose.

This food contains, therefore, only a very small amount of invertible substance, but the pancreatic ferment appears to be still active, though by following the directions the full advantage of this is not obtained. When merely heated for thirty minutes we get 25·6 per cent of glucose, but when boiled it forms only 20 per cent. This food would thus yield a more assimilable product if it were simply heated and not boiled. The starch is either unchanged or present as soluble starch when made according to the directions, but by heating it for long a large amount becomes dextrinized. Owing to the presence of the dried milk, however, this cannot be done, for then the pancreatic ferment would act for too long a time, and render it bitter. Thus the presence of milk in this food does not allow of us obtaining the full advantage of the amylopsin, and, besides this, the milk is predigested, and this is unnecessary in a food designed generally for the use of infants. This food would be valuable in acute diseases.

3. *A Non-Farinaceous Food* (M. F.).—This is in the form of a yellow powder possessing a sweet malt taste. It dissolves in water, giving a muddy yellow solution, and possessing a neutral reaction.

It is directed to be prepared by dissolving the food in cold water, then adding milk and water, and heating gently.

Having made a 2 per cent. solution, I examined it.

(1.) Prepared by dissolving in warm water. There is no unchanged starch present. No coloration results from the addition of iodine, so that any starch originally present has been converted into achroodextrins, maltose, or glucose. Glucose forms 29·8 per cent. of the food.

(2.) When acidified and heated for two hours, glucose forms 30·4 per cent. This is almost exactly the same as before, and so this food contains no invertible substance.

(3.) When heated alone for thirty minutes the same results were obtained.

The amount of reducing substance is the same after simply dissolving the powder as after heating with the addition of an acid. There is thus no cane sugar or other easily invertible substance present. The slight increase in the amount of glucose is probably due to conversion of some non-reducing dextrins into those capable of reducing copper from its solution, through the prolonged action of heat. Any starch which was originally present has been during manufacture converted into achroodextrins, maltose, or glucose.

This food contains, therefore, carbohydrates in their most easily assimilable condition.

4. *The same, with Desiccated Milk added* (M. L. G.).—This is in the form of a brownish-yellow powder having a sweetish taste of malt. It dissolves in cold water, forming a turbid fluid, and has an alkaline reaction.

It is prepared by dissolving in warm water. A 2 per cent. solution was made with warm water.

(1.) Solution made by heating only. Small amount of erythrodextrin present. Glucose forms 32·7 per cent.

(2.) After being acidified and heated for two hours, it gives no reaction with a solution of iodine. Glucose forms 33·6 per cent.

This is almost exactly the same as before inversion. There is, therefore, only a trace of invertible substance present, or rather, and more probably, convertible substance, for the erythrodextrins have become achroodextrins and reducing dextrins, so increasing the glucose nearly 1 per cent.

This food closely resembles the preceding in its composition, containing in addition desiccated milk. The carbohydrates are all in a very easily assimilable condition.

5. *A Pancreatized Food* (B. F.).—This is a dry white powder, resembling wheaten flour.

In preparing it for the use of infants, we are directed to mix it into a paste with one-third cold milk, then to add two-thirds boiling milk or milk and water, and set it aside in a warm place. In fifteen minutes it will have been sufficiently digested, and should then be slowly heated till it boils, when it is ready for use.

(1.) Mixed with cold water it has a slightly alkaline reaction, gives the starch reaction with iodine, and shows a trace of erythrodextrin. It causes no reduction when boiled with Fehling's solution.

(2.) A 2 per cent. solution made with hot water, and examined at once, shows starch abundantly; erythrodextrin in greater amount than when made with cold water. Glucose is present to 7·8 per cent.

(3.) Prepared according to the directions; kept warm for a quarter of an hour, and then boiled. Starch reaction is not so marked; soluble starch and erythrodextrin in much larger amounts, and glucose forms 17·2 per cent.

(4.) A solution prepared according to the directions and heated, after being acidified, for two hours, shows only a very small amount of starch to be present. Much erythrodextrin is present, and glucose 13·9 per cent.

After this prolonged heating, the amount of reducing material has diminished. I do not know how to account for this, unless the pancreatic ferment has split up the products of digestion during the lengthened time during which it has acted. I repeated this and similar experiments several times, but always with the same result. This would seem to prove that there is a ferment,

as has already been described, in the pancreas which is destructive to grape sugar.

This food contains, therefore, in its original state no substances which have a reducing action. It has undergone no previous treatment with malt. During its preparation, however, the pancreatic ferment which it contains acts on the starch, and in this way we have a large amount of dextrin and glucose formed, though, even after all, there is yet a small amount of unchanged starch left unacted on. The food seems to consist of flour chiefly, mixed with some pancreatic ferment. When milk is used in the preparation of this food it also, of necessity, will undergo digestion by the same ferment.

6. *A Food designed for the use of Infants during the first Three Months.*—This occurs as a gritty yellow powder having a slight cheesy smell, and with a sweet milky taste. It leaves a saline sensation in the mouth.

It is prepared by dissolving to a smooth paste in hot water, then adding a sufficiency of hot water.

(1.) Mixed with cold water it gives no reaction with iodine, and contains 34·7 per cent. of reducing sugar (glucose).

(2.) Made according to the directions, there is no change from the preceding.

(3.) Made with warm water and kept at 38° C. for thirty minutes, still same results.

(4.) Heated on water bath for two hours after acidification, the sugar has increased to 44·6 per cent.

This food contains, therefore, no unchanged or soluble starch, or even dextrins capable of higher transformation. Nor does it contain erythrodextrin. It seems to be what is affirmed, dried cow's milk from which has been abstracted the excess of casein; while cream, soluble albumin, and milk sugar have been added, to make it resemble ordinary mother's milk. The increase in the amount of sugar seen after heating with acid is probably due to the splitting up of lactose into glucose and galactose.

7. *A Food for Infants up to their Seventh Month.*—This is also in the form of a fine yellow gritty powder with sweet milky taste. It also leaves a saline taste in the mouth. It possesses a faint odour of cheese and malt.

It is prepared by mixing with hot water and then adding a sufficiency of hot or boiling water.

(1.) A cold extract gives no reaction for starch. It contains 36·2 per cent. of sugar (as glucose).

(2.) Prepared according to the directions, the constituents remain in the same proportion.

(3.) Prepared with warm water, and kept for thirty minutes at 38° C., the sugar has increased to 39·05 per cent.



(4.) Heated on water bath for two hours after acidification, the sugar now amounts to 39·65 per cent.

There is no starch of any kind present, but only dextrans and sugar. There is, however, some malt present, as the amount of sugar has increased during the period of simple heating. Some of the lower non-reducing dextrans have been changed into higher and reducing dextrans or into sugar through the action of this malt. This food thus resembles the preceding, except that it possesses originally more sugar, dextrans, and malt.

8. *A Malted Food* (A. H. F.)—This appears as a cream-coloured powder, having a sweet taste of flour and malt.

It is prepared by adding boiling milk and water to the food, which has previously been mixed to a paste with cold water, and to which cane sugar has been added.

(1.) Mixed with cold water it shows the presence of unchanged starch, but no soluble starch, and contains 10·85 per cent. of reducing substance.

(2.) Made according to directions, and examined at once. Unchanged starch is present along with erythro-dextrin, and reducing substances form 12·4 per cent.

(3.) Prepared as directed, and kept warm for thirty minutes. Trace of unchanged starch. No soluble starch, or mere trace. Erythro-dextrin in large amount, along with achroo-dextrans. Reducing substances form 12·85 per cent.

(4.) Heated for two hours on water bath after acidification. Shows the presence of trace of unchanged starch. Soluble starch. Much erythro-dextrin. Reducing substances, 14 per cent.

These three latter preparations are intended to form a continuous diet for the infant from birth up to the end of the first year.

The first (No. 6) consists merely of ordinary cow's milk so treated as to resemble closely human milk. It is then sterilized and dried in vacuo.

The second (No. 7) consists in an addition to the first of maltose, dextrans and malt, together with soluble salts.

While the third (No. 8) consists of starch which has undergone a partial conversion through the action of malt. During the process of preparation the degree of conversion is still further advanced.

The *first food* is designed for infants up to three months of age, the *second* from three to seven months, and the *third* (along with ordinary cow's milk) from this age onwards.

9. *A Malt Food for Infants* (S. M. F.)—This is a fine cream-coloured powder, closely resembling and having the taste of heated flour. Directions for preparing the food: It is to be mixed with cold milk, or milk and water, into a thin paste. Then boiling milk or milk and water is to be added till the food thickens (at 140° F.). It then rapidly becomes fluid, and is ready for use.

(1.) When mixed with cold water its reaction is faintly alkaline, and it shows the presence of unchanged starch, but no sugar.

(2.) A 2 per cent. solution prepared according to the directions, but with water only. Starch is present in small amount. Much erythrodextrin and achroodextrin. Glucose 9.25 per cent.

(3.) Part of this solution kept at 100° F. for thirty minutes. Starch in very small amount. Much soluble starch and erythrodextrin. Glucose 11.1 per cent.

(4.) A solution acidified and heated at 140° F. for two hours shows only a trace of unchanged and soluble starch, erythrodextrin in very large amount, as also achroodextrin. Glucose amounts to 15.6 per cent.

(5.) A solution prepared by adding the food to tepid water, and keeping it at 80° F. for thirty minutes, showed very little unchanged or soluble starch, much erythro- and achroodextrin. Glucose forms 5.3 per cent.

This is clearly one of the malt foods, consisting of flour and malt. Under the influence of heat and solution, the latter acts on the former to cause its conversion into dextrins, maltose, and glucose. We see that the conversion is more complete the longer the mixture is kept warm. If, however, the temperature is never raised high (as in 5), the diastase of the malt converts the starch merely into dextrins, and but little reaches the condition of sugar. If kept for long, however, even at this temperature, all the starch ultimately becomes changed into maltose and glucose.

10. *A Self-Digesting Whole-Meal Food.*—This is a fine yellowish powder with brown particles in it, and possessing a strong taste of malt. It is cooked in exactly the same way as the preceding.

(1.) Mixed with cold water, its reaction is faintly alkaline, and it consists solely of unchanged starch with the merest trace of sugar.

(2.) Prepared according to directions. Much erythrodextrin, achroodextrin, soluble starch, and unchanged starch. Reducing substances form 7.35 per cent.

(3.) When kept at 120° F. for thirty minutes the erythro- and achroodextrins have increased at the expense of the starches, and reducing substances form 8.3 per cent.

(4.) Acidified and heated for two hours. Still more dextrins present, and reducing substances 11 per cent.

This food does not differ from the preceding except that apparently whole meal has been used instead of flour.

11. *A Malted Food (H. M. F.)*.—This is a granular, gritty yellow powder, having a sweet milky taste.

It is prepared simply by dissolving in hot water. The amount of the powder used is regulated by the age and condition of the child.

(1.) Five per cent. solution made with warm water. Alkaline reaction. There is no unchanged or soluble starch, and no erythro-dextrin. No precipitate is formed on adding acetic acid. Glucose amounts to 23·25 per cent. of the food.

(2.) Part of this solution kept at 100° F. for thirty minutes showed an increase of the glucose to 25 per cent.

(3.) Acidified and heated for two hours. Glucose forms 31·25 per cent.

This preparation is said to consist of desiccated cows' milk, malted flour, and alkaline carbonates to neutralize the acidity of the milk.

It contains no starch or early-formed dextrins. It has been almost completely malted already. The length of time it is heated by itself increases but little the amount of sugar. The increase in reducing substance by the prolonged heating with acidification is not likely due to added cane sugar, but most probably to the more complete conversion of some of the higher dextrins (achroodextrins) into reducing dextrins and sugars.

This preparation contains, therefore, dextrins, maltose, glucose, albuminous materials, and mineral salts.

12. *A Patent Cooked Food for Infants* (R. F.)—This is a cream-coloured powder, looking and tasting like heated flour. Directions: Mix the food with water or milk to form a cream; add hot water or milk, "stirring briskly while boiling." It is then ready.

(1.) Prepared with cold water it has a faint acidity, and consists of unchanged starch with no sugar.

(2.) Prepared according to directions, but not boiled, only kept at 120° F. for thirty minutes. It gives only unchanged starch reaction, along with the faintest trace of reducing sugar.

(3.) Prepared by boiling—starch alone is present; no sugar. Even if kept for thirty minutes at 120°, no further change results.

(4.) Acidified and heated at 140° F. for two hours, a large amount of erythro-dextrin is present, along with soluble and unchanged starch. Glucose forms 5·4 per cent. of the food.

This food contains apparently only flour; and if prepared according to the directions, we only get a paste containing no dextrins nor sugar. At best the starch is only present in its soluble form. Prolonged heating after acidification converts a good deal of the starch into dextrins and into a small amount of sugar.

13. *A Farinaceous Food* (F. N.)—This appears in the form of a light cream-coloured powder, with a taste of heated flour. Directions: Mix the food with cold water to form a thin paste; add boiling water, and boil gently for five or seven minutes. Then milk and sugar are added, and it is ready.

(1.) Mixed with cold water it has a neutral reaction, contains unchanged starch and no sugar.

(2.) A 2 per cent. solution made as directed, but not raised above a temperature of 140° F., and kept at this for half an hour, shows only the presence of unchanged starch. Glucose forms 1.65 per cent.

(3.) Prepared according to the directions, no sugar or dextrans, but only unchanged starch.

(4.) Part of this solution (No. 3) kept at 100° F. for thirty minutes shows a trace of sugar.

(5.) Acidified and heated for two hours. Nearly all the starch has been converted, only a trace being left unchanged. Erythro-dextrin is present in large amount. Glucose forms 6.88 per cent. of the food.

This food is very similar to the last. Prepared in its usual way, the starch remains almost unchanged. If heated for long, however, a large amount of conversion takes place, and more especially if rendered acid.

14. *Another Farinaceous Food* (F. H.)—This is a fine powder, closely resembling ordinary flour. It is prepared by mixing with a little cold water. Boiling water is then added, and it is boiled for eight minutes. Milk and sugar are added to make it agreeable.

(1.) Mixed with cold water it has a neutral reaction; contains no sugar or dextrin, but only unchanged starch.

(2.) A solution made with warm water and kept at 140° F. for thirty minutes, but not boiled, shows the presence of dextrin in small amount, much unchanged starch, and glucose forms 2.27 per cent.

(3.) Prepared as directed, only unchanged starch is present; no sugar or dextrans.

(4.) Boiled and kept warm for thirty minutes, gives results similar to No. 3.

(5.) Acidified and heated for two hours, shows very little unchanged starch, much erythro-dextrin and achroodextrin. Glucose forms 6.25 per cent. of the food.

This food also closely resembles the two preceding. If made as indicated we get only a flour paste, but if heated for long a large amount of conversion results.

15. *Another Farinaceous Food* (F. N. H.)—This appears as a coarse white powder, with which are mixed small hard brown scales like bran. For infants' use it is prepared by pouring equal parts of boiling milk and water over the food which has previously been slightly moistened. It then is boiled for five minutes.

(1.) Mixed with cold water it has a faintly acid reaction, and shows only the presence of unchanged starch.

(2.) A solution prepared with hot water and kept at 120° F. for

thirty minutes shows much unchanged starch; small amounts of dextrins. Glucose forms 5·2 per cent.

(3.) Prepared according to the instructions, only unchanged starch; no sugar or dextrins.

(4.) Part of the latter mixture heated for thirty minutes shows a trace of sugar.

(5.) Acidified and heated for two hours, much unchanged and soluble starch; erythrodextrin in small amount; glucose 2·32 per cent. of the food.

It is evident that the boiling which the food is directed to have destroys the small amount of power of the converting ferment which it seems to possess. When simply made with warm water and kept warm, it develops 5·2 per cent. of glucose, whereas if boiled there is none.

#### *General Conclusions regarding Infants' Foods.*

We gather from the preceding analyses of the principal varieties of infants' foods the following:—

1. Most of these consist of wheaten flour mixed with malt or extract of malt. The latter is supposed to act on the starch of the flour during the process of cooking, and by the diastase which it contains to convert the starch into maltose and glucose.

I have shown, however, that in several of these varieties, if prepared according to the directions accompanying each, only a very small conversion of starch occurs. A temperature of 140°-150° F. is most suitable for the action of diastase; while if carried to 212° F. the ferment is killed or its action arrested. Now, several of these foods are prepared by adding boiling milk or water, and then boiling for from five to ten minutes. Such treatment effectually prevents the starch from undergoing any conversion, or soon brings to an end any that is going on already. I have shown that if some of these foods be prepared by adding warm water, and be then kept warm for half an hour, the temperature not being allowed to rise much above 150° F., part of the starch undergoes conversion, and we then find dextrin, maltose, and glucose, with or without unchanged starch, depending on the time allowed and on the strength of the ferment.

Some of the foods composed of flour and malt are directed to be prepared thus, and with these no fault can be found. Those, however, which are directed to be boiled, I have no hesitation in saying, are quite unsuitable as foods for young infants. Not that the food is in itself bad, but owing to the mode of preparation which renders it so. If properly cooked, some of these would make fairly good foods.

Those containing ground malt should always be used in preference to those which contain the extract, as the former is much more active in its converting power.

It is to the mixtures of malt and flour that I look to the greatest improvement in the feeding of infants. By varying the time during which such mixtures are heated, we may convert the starch to any degree we desire, and so make it suitable for the child at different ages or according to its condition of health. We may either wholly convert the starch by prolonged heating, so making it suitable as a food in addition to milk for young infants; or the conversion may be only partial, leaving still soluble starch, dextrins, and maltose. As the child grows older the amount of conversion is proportionally lessened, till when it has arrived at a suitable age or condition unchanged starch may be given alone.

In a few of these foods cane sugar is also added; but this is an addition to infants' or invalids' food which I have elsewhere sufficiently condemned.

2. Instead of malt, some contain the pancreatic ferments. These act both on the starch and on the milk with which the foods are prepared, and so both are predigested.

Such foods must be most valuable in conditions of great debility, inanition, or exhaustion in infants or invalids. In an ordinary food for children, however, we do not wish to digest the milk, which is the natural food of the child, and which usually can be digested well enough. Proteids are generally well digested by infants, and we take advantage of this to feed them on meat infusions when there is great debility. As a general rule, therefore, we do not desire to predigest the proteids of milk, but only the carbohydrate element which the infant cannot properly, or only partially, digest. It has been shown that kittens fed on fully predigested milk did not, for a time, thrive so well when this was replaced by only partially predigested milk as their brothers and sisters who were fed on ordinary milk. The use of predigested foods lessens the activity of the glands which ought to secrete the digestive fluids. We ought not, therefore, to give artificially digested food for a longer time than is absolutely necessary.

3. In some of the foods the starch has been converted previously through the action of the diastase of malt. In these we find no unchanged starch, or at most mere traces of it; erythrodextrins and achroodextrins are found in varying amount; while maltose and glucose occur usually in large amount.

Nearly all of these are made from flour, and so contain, besides, the vegetable albumins and mineral matters. In some foods an alkaline carbonate is added in order to neutralize the acidity of ordinary cow's milk.

Such foods as these are very easily absorbed. The starch is almost wholly changed into easily absorbable dextrins and glucose, and so they require little or no digestion. This is manifestly of the greatest importance to the child, in whom the power of digesting carbohydrates is at the minimum.

4. Combinations of dextrins and starch are often met with, and

are highly vaunted as valuable foods for infants. Such foods consist simply of flour which has been subjected to a high temperature, and has thus been baked. The starch during this heating becomes changed into soluble dextrins. If the flour has been carefully heated, and for a long enough time, the starch becomes wholly dextrinized. In the usual foods, however, such treatment has rarely been carried sufficiently far; thus only a part, and usually a small part, of the starch has undergone conversion even into the early formed or low dextrins. They contain usually much unchanged starch, along with the albuminous constituents and salts of the grain. Domestically this is known as the "flour-ball," and is prepared by boiling flour in a cloth for about twenty-four hours. It then forms a hard ball, which, after the translucent outside skin has been pared off, consists of a dense white substance, made up of dextrinized starch. This is then ground down and mixed with milk for the infant's use. Used in this manner it forms an easily assimilated carbohydrate, and, acting as a mechanical diluent, helps to prevent the milk from coagulating in large or firm clots in the stomach.

If these dextrinized foods are thoroughly well prepared they form valuable additions to the milk. If imperfectly dextrinized, however, the large amount of unchanged starch which is also given forms a great drawback to their use as safe articles of diet.

5. Many preparations sold as food for infants consist simply of flour or unchanged starchy matter.

Such foods ought never to be given, as only a small part can be digested by the infant. We find that many of these, besides, are very coarsely prepared, and show, when examined by the microscope, the presence of husks, spiculæ, etc., which must prove most irritating to the delicate intestinal mucous membrane, and which form a sufficient cause in many cases for the diarrhoea which ensues after such food is administered.

Owing to the relatively small number of glands in the intestinal tract in the young child, and to the immaturity of those which are present, the infant is unable to assimilate carbohydrates which require extensive change.

As the child grows older greater liberties may be taken with his digestion; but at an early period of his life it is culpable ignorance to tamper with the delicate machinery of digestion by giving any or the first food which may present itself to the parents or to the seller.

It seems to me that the general and unrestricted sale of such preparations as food for infants ought to be prohibited; or that, at least, they should only be sold by qualified chemists who would first inquire as to the age and condition of the child, and who would thus know whether the food were suitable or not. A still safer plan would be that none of these foods should be supplied unless prescribed by a medical practitioner,—in fact, dispensed in

the usual manner in which drugs are sold. In order to do this, however, the method of manufacture and composition of each food, both before and after being cooked, would require to be made public and printed on each packet.

It is the wholesale vending, nay, the intrusion, of such foods on people that renders their use so general. It is a fact that so soon as a birth is advertised in the newspapers, many of the manufacturers of these foods send samples of their preparations to the parents, and, of course, each is accompanied by its own laudatory literature. The parents, knowing little regarding the proper feeding of their child, select one which they think is best, and so, unguided by experience and ignorant of physiological processes, they usually choose that which is most highly vaunted or is accompanied by portraits of the fattest babies reared on such food.

It is on account of the incalculable harm which is thus done—unwittingly in many cases—to the infantile population, that I would put the sale of such foods under a restriction. For this purpose a special clause might be introduced into the "Sale of Foods and Drugs Act," so as to make the indiscriminate sale of infants' foods penal.

Almost all the forms of infants' foods are directed to be made with milk or with a mixture of milk and water, except those which already contain desiccated milk.

In those which contain malt the starch becomes converted in large part during the preparation of the food. Now it has been shown that if malt be added to milk, and if this mixture be kept at 100° F. for some time, the casein of the milk undergoes such a change that it becomes uncoagulable by acids. I have verified this, and find that when acetic acid is added to milk which has been heated with malt no curdling takes place. This fact must be of great importance as regards the digestion of milk, for one great cause of indigestion is the formation of large clots of milk in the stomach. In foods containing malt the milk is also acted on during the process of cooking, and so rendered uncoagulable by the acid of the stomach. Might this not prove serviceable in the treatment of some forms of indigestion following the use of cow's milk? Instead of mixing the milk with any starchy food, would it not be preferable to keep it in contact with a solution of malt for some time so as to prevent the formation of dense coagula in the stomach?

Such foods containing malt, when the cooking causes an almost complete conversion of starch, or those which are already entirely converted, when added to diluted cow's milk, and with a little cream added (for nearly all these foods are deficient in fat, and the dilution of the milk reduces the percentage of fat much too far), make, in my opinion, the most suitable substitute for mother's milk, or for infants with whom ordinary cow's milk does not agree, or who are not thriving on it.



Not only are these foods good as adjuvants to milk, but they are sometimes the only food which the child can digest. After once that fermentative changes have taken place in the gastric and intestinal contents, with consequent vomiting and diarrhœa, the bacteria which have caused these remain, and cause milk to become acid as soon almost as it has been taken. Under such circumstances the use of milk must be stopped, and we have then recourse to such foods as the above, along with infusions of meat, as veal or chicken broth.

At a later date also they are of great value, when we are beginning to add other food materials to the milk. What better can we begin with than some of the easily digested carbohydrates ?

It would be improper in me to specify by name the particular foods which I would recommend in feeding infants of various ages. After what I have said, however, there can be little difficulty in choosing the particular food for the particular age and condition of the child. I have shown what I consider to be the most easily assimilated forms of the carbohydrate group, and from the analyses of the carbohydrate constituents of the most widely-known foods for infants now on the market which I have given, it will be easy to select those which would form desirable additions to, or substitutes for, a purely milk diet.

What I have stated regarding the kind of carbohydrate food which may be given to the infant applies, and in some cases with even greater force, to the dyspeptic and invalid, or during senescence, when the digestive organs are feeble and assimilation is less active than formerly.

In such diseases as chronic gastric catarrh, dilatation of stomach with concomitant atrophy of the gastric glands, acid dyspepsia, cancer of stomach, congestion of the gastric and intestinal mucous membrane from heart or lung disease, I would forbid the use of ordinary farinaceous food, unless in very small amount; while I would advocate the use of partially or completely converted starch. In ascites or dropsy such food would act most beneficially and even therapeutically, for glucose and even dextrins form powerful diuretics, and act without raising the blood pressure.

In many febrile conditions the amylolytic action of the salivary and pancreatic secretions is greatly lessened, and only harm can be done by the administration of ordinary starchy foods; whereas if predigested they act as valuable and easily assimilated foods. Again, in those cases where there is great deficiency of hydrochloric acid in the gastric juice, as, for example, in anæmia, simple and pernicious, cancer of the stomach, etc., where proteid matters are not digested well, starchy food ought to bulk largely in the diet, as the amylolytic action of the saliva continues for long in the stomach, there being little or no acidity to cause its cessation.

The carbohydrates, when either partially, or specially when wholly converted, form easily assimilated articles of diet, and give

rise to no inconvenience, unless when, like any other food, they are taken in excess. They are quickly absorbed, and thus form a most valuable addition to our fat-producing or energy-saving foods.

It is by attending to such points as I have drawn attention to in managing the dietary of infants and invalids, along with an improved hygiene, that we may in the future with confidence expect a greatly diminished rate in infantile mortality, and a much improved condition in the general health amongst the new members of our population.

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*The President* said they were much obliged to Dr Robertson for this important contribution on infantile dietetics, and they should be glad to hear remarks on it from those well qualified to judge of it from practical experience. He thought they might call on Dr Carmichael to express his opinion.

*Dr James Carmichael* said he must bow to the ruling of the chair, but he was under the disadvantage of not having heard all Dr Robertson's paper, so that he could not criticise it. Evidently it was such a long dissertation, and went in such detail into this matter, that one would require to read it in order to discuss it. What the President, perhaps, desired was that he should express in general terms his opinion of these infant foods. He did not quite gather from Dr Robertson's paper whether he recommended them as substitutes for mother's milk during the lactation period, which the makers advertised them to be. This, of course, he could not suppose for one moment that Dr Aitchison Robertson would do, nor would any medical man who had given attention to the subject, and who knew and understood the physiology of infantile dietetics. He thought that they must altogether discard these foods as substitutes for mother's milk during the lactation period,—*i.e.*, during the first period of life, because from a physiological and chemical point of view they were totally unsuited. They did not contain all the elements of nutrition necessary for the child. Among the foods which Dr Robertson had alluded to there was only one that contained all the elements of nutrition,—*viz.*, malted milk. The others were deficient in one of the most nourishing ingredients,—*viz.*, fat. Now, the most important of these elements in milk, the typical food, were certainly the proteids and the fats in addition to the carbohydrates. In regard to these foods generally, many of them were cooked, or at all events altered, during preparation, which deprived them of the fresh quality of a natural food. Being more or less cooked, they seemed to act on the child very much in the same way as cooked or salted food did on the adult; they did not possess that which most fresh foods, especially vegetable foods, contained, the antiscorbutic property. Now they were beginning to find out that in early life they must pay great attention to this in the feeding of children. If they fed them on such foods, they were

apt to develop not only rickets, but also scorbutic symptoms, which could be prevented by suitable change of diet. He had referred to these foods as professed substitutes during the lactation period for the mother's milk or fresh cow's milk, and he had only done so wholly to condemn them. But they had not only to feed the child during this critical period artificially, but afterwards, when dentition was fairly advanced, they had to feed it like a grown-up person, on a mixed and varied diet, because they must always bear in mind that children were little men and women, and had their peculiarities of digestion in regard to food, and they must study each child and vary the food according to the nature of the case. When the child was weaned there was no doubt that these foods, if taken along with other foods, were suitable and useful. Any of these farinaceous foods combined with fresh milk formed useful nutriment for a child just after weaning, given in the form of thick gruel or porridge. But they were not so good as the natural foods themselves. He much preferred the natural farinacea,—wheat, barley, or oats. Bread was the staff of life, so was wheat flour. Barley flour was equally nutritious, oat flour even more so. So cooked in the ordinary way, they were a long way more useful than any of the artificial foods. As to the diastatic property of the prepared foods, he thought it was no advantage, perhaps the reverse, to healthy children. He would confine the use of partially predigested foods to cases of disease where digestion was very much weakened, as in chronic gastro-intestinal catarrh. But he for one—and he knew it was the opinion of many of his professional brethren with whom he had spoken on the subject—maintained that they were not to be compared with the ordinary natural foods as nutriments. A child as it grew older could convert starch into dextrin in the ordinary way, and it was an insult to its natural digestive powers to give it predigested food. He must apologise for the discursive nature of his remarks. These were some of the principal points he had thought it well to refer to.

*Dr W. T. Black* said he should like to throw out the suggestion whether the carbohydrates in the form of sugar would not have some antiseptic power. Would sugar not have the same effect internally as externally? He would also ask whether *Dr Robertson* would not justify a diet of porridge? He was unable to say whether he (*Dr Robertson*) regarded it as founded on scientific principles. Its justification was from its use and results. The scientific exposition of it might be rather an interesting curiosity.

*Dr Lockhart Gillespie* said he had listened to this paper with much interest, because lately he had been looking into part of this subject himself. He did not know that he had heard anything very new, but the facts had all been put quite plainly before them. A French physician, *Dr Brouget*, at the beginning of this century expressed the opinion that mother's milk was the

worst thing children could take, because it bred immorality and hereditary disease. Three centuries before that a chemist, Van Helmont, held that mother's milk was exceedingly bad, because it led to inborn propensities being transmitted. He recommended bread boiled in beer or honey as infants' food. He (Dr Gillespie) did not know whether it was worth while analysing that. One or two points he had noted. Dr Robertson said that gastric digestion was the same in children as in adults. He (Dr Gillespie) had an idea that in infants the ferment rennet was very much stronger. There was a much larger quantity of *lab*, as the Germans called it, or rennet. Dr Robertson had said nothing about want of fat in condensed milks. He might refer to the *British Medical Journal* of last summer or the summer before, where an account of a large number of analyses of condensed milks was given. Only one or two were found to have been made from ordinary milk. Most were made of skim milk evaporated down and with sugar added, generally cane sugar. A third set were condensed milk with some fat, lard, or suet added in small quantity. One of the causes of illness in children was want of fat. They might give children any amount of phosphates and iron. If they had not enough fats and proteids they would suffer. One reason for giving these foods was, according to Dr Robertson, that they rendered the curd more digestible. Arthur and Page found that all that was required was to add a small quantity of a lime salt to the cow's milk. Two special foods he would like to mention—Horlick's and Muffler's. The latter was, he thought, the best. It was made by the Aylesbury Dairy Company, and consisted of desiccated milk with powdered wheaten flour, powdered white of egg, and a little lactose. Another thing that Dr Robertson might have mentioned was humanized milk of the Aylesbury Dairy Company. No cane sugar was added. It was all lactose, which, of course, did not cause anything like the same irritation of the intestinal canal. With regard to oat flour Dr Carmichael had taken the words out of his mouth. A little of Scott's oat flour added to the milk gave better results than anything else. The child fed on mother's milk did not take much at a time, though able to digest a good deal. When fed artificially it was often made to take a fixed and too large a quantity. In all artificial feeding there should be a text hung up on the wall of the nursery with the words "little and often."

*Dr Allan Jamieson* said that at one time he devoted a considerable amount of attention to infants' food, and he could support a great deal of what was said by Dr Gillespie and Dr Carmichael. One or two things had struck him at that time, and struck him still. One was that mothers were apt to give the milk too dilute. They were afraid, too, of adding much lime-water. They seemed to think it very dangerous and potent, and instead of putting in a quarter or a sixth of lime-water, they

added a teaspoonful to an occasional bottle, and used water instead. Consequently the milk was not prevented from forming a hard coagulum in the stomach. Another thing was that they gave ordinary food often too hot, and that, he thought, was the reason for the food being so frequently rejected; not that he would recommend that bottles which had a thermometer attached should be employed, because in one or two cases disastrous accidents had arisen from the thermometer getting broken and some of the mercury being swallowed. As to the advantages of some of the well-known substitutes, two things that he knew of formed the best addition to the food when the child had reached the teething period. One of these was known as "Chapman's Entire Wheaten Flour." They must have the entire wheaten flour. It would not do to have the finest material ground down and prepared. They must have the whole ground (only the husk being rejected) in order to get the salts. The advantage of oat flour was that it contained fat. It was one of the richest of the cereals in fat. It was the presence of fat which had made it such a nutritious food for the Scottish nation, now far too much replaced by tea, also for children as a substitute for the natural food.

*Dr Burn Murdoch* said he had listened with interest to this paper, which was a *résumé* of a subject which, he thought, they were the better of being told something of now and then, also to several of the speakers, notably to the practical remarks of Dr Carmichael and Dr Gillespie. He could not help feeling that there was a great deal of nonsense talked about the difficulty of feeding infants. He had not found that difficulty, if the milk was properly prepared. He had rarely in private practice to fall back on artificial foods. Analyses of these foods were enough to show thinking men that they resembled neither human milk nor good cow's milk. One point he wished to emphasize more strongly than Dr Gillespie had done, viz., that the amount of food required by a young infant was exceedingly small. It had been fully worked up, and the statistics were available for those who chose to read them in the books,—14, 15, or 20 ozs. in twenty-four hours during the first month of life, and not more than 30 or 40 ozs. of fluid in the twenty-four hours, up to the end of the first year,—showing the value of mother's milk. Those who were in family practice had only to contrast with that, the vast amount of fluid which a thoughtless mother would pour through an unfortunate infant. Not very long since he found a lady, who was daily giving her child over 80 ozs. of fluid in twenty-four hours. He only succeeded in bringing home the folly of such excessive feeding after showing her four quart bottles full of water. With regard to grains, he could entirely corroborate what Dr Carmichael and Dr Gillespie had said as to the great importance of choosing the proper grains for thickening both milk foods and soups. He always preferred the three home grains.

*Dr James Ritchie* said he thanked *Dr Robertson* for his paper, because mainly it seemed to be an argument for the use of natural foods, first of all of mother's milk, next of cow's milk, in preference to any of these artificially prepared foods. He (*Dr Ritchie*) agreed entirely with what *Dr Carmichael* had said, that if they could use any of the natural grains, such as wheat, oats, barley, they were preferable. In a town he believed in sterilized milk. He agreed with *Dr Carmichael* that cooked milk did not yield so much nourishment as uncooked. It had been demonstrated that boiled milk was not so readily absorbed as uncooked milk. But as in towns milk was produced under somewhat unfavourable conditions, it was wise to give children sterilized humanized milk. If that could not be done, then let them mix with water, cream, and sugar, and add a certain amount of flour prepared in the well-known method of the flour-ball, or instruct parents to get some flour cooked in the oven by their baker. The flour was cooked in order to have the starch transformed, and the object of giving it, he explained to parents, was not to give nutriment, but to make a soft open curd by its mechanical presence. He dared say some of the members would remember that a number of years ago he had the honour of reading a paper before the Obstetrical Society, in which he described a good many experiments he had made in preparing foods for children and digesting them out of the stomach. He had always had the idea before these experiments that lime was most useful, and gave lime-water in large quantities where there was no constipation, but since then he almost never gave it except in diarrhoea. When he digested the food out of the stomach and added lime-water, he got a beautiful curd in small soft flakes resembling that of mother's milk. But the secretion of the stomach was acid; it neutralized the lime, and when this was done they got almost as hard a curd, in fact quite as hard a curd as they got with plain water. The more the milk was diluted with water the softer was the curd. (*Dr Gillespie*—"I referred to salts of lime, not to lime itself.") *Dr Ritchie* apologised for his mistake. Continuing, he said that *Dr Robertson* should send his paper to the makers of these foods, so that they might be able to modify the instructions they sent out, which had the effect of spoiling the foods in their preparation. Sometimes malt food did good; in other cases it was absolutely useless.

*Dr Carmichael* said he would strongly recommend *Dr Robertson* not to follow *Dr Ritchie's* advice, because he would not get thanks, perhaps the reverse. When he (*Dr Carmichael*) was writing a little book for *Mr Pentland* the publisher, he on two occasions mentioned the names of these foods, and *Mr Pentland* insisted that he should take it out. Otherwise he might get himself into serious trouble.

*Dr Aitchison Robertson*, in reply, said he thanked them for the

way in which they had received his paper. Some appeared to have misunderstood his object, which was not to formulate a proper dietary for children, but to give a *résumé* of the constitution of infants' foods as they occurred in the market. He did not wish to lay down any hard and fast rules. The mother's milk was in general the best. But certainly it was the worst food if the mother was unhealthy herself. In that he agreed with the Frenchman mentioned by Dr Gillespie. He entirely concurred with the other speakers, that most of these foods were unsuitable for children. At the same time, these foods were very largely employed, and he had tried to show in what circumstances they might be safely or even advantageously used. He emphasized the use of sterilized cow's milk as the next best to healthy mother's milk. Dr Black had asked him whether sugar might act as a preservative. He (Dr Robertson) thought that in the dilute form it would rather decompose. Porridge made of oat flour was no doubt an excellent food for children of a reasonable age when able to digest starch.

## 2. ENORMOUS PRIMARY SARCOMA OF THE LIVER, IN WHICH A LARGE BLOOD CYST, SIMULATING A HEPATIC ABSCESS, WAS DEVELOPED.

By BYROM BRAMWELL, M.D., F.R.C.P. Edin., F.R.S.E., Assistant Physician, Royal Infirmary; Lecturer on Practice of Medicine, School of Medicine.

With Pathological and Microscopical Report by R. F. C. LEITH, M.D., F.R.C.P. Ed., Pathologist, Royal Infirmary; Lecturer on Pathology, School of Medicine.

THE following case presents many points of pathological and clinical interest. The more important pathological features are:— (1) The rarity of primary sarcoma of the liver; (2) the large size of the tumour; (3) the fact that a large blood cyst (the cavity of which contained 53 ounces of anchovy-paste-coloured fluid) was developed in the interior of the tumour.

Clinically, the case is chiefly interesting from a diagnostic point of view. The physical signs exactly simulated those of an abscess of the liver. The liver was greatly enlarged; the dulness extended chiefly in the upward direction; the upper limit of the dulness was a curved line corresponding to the S-shaped curved line of a moderately large pleuritic effusion; the apex beat of the heart was displaced upwards and to the left; the right side in the infra-axillary and right hypochondriac regions was enlarged and bulging; a tender spot was present in the sixth right interspace in the anterior axillary line (indicative of the "pointing" of the fluid collection); an aspirator needle introduced into this spot entered a large cavity, and from this cavity 53 ounces of anchovy-paste-coloured fluid (highly suggestive of an abscess of the liver) were withdrawn on aspiration.

But against the diagnosis of hepatic abscess were the facts that there was no history of dysentery or other cause of hepatic abscess; and that the fluid removed from the cavity in the liver contained a much larger quantity of blood (red blood corpuscles) and a much smaller quantity of pus (leucocytes or pus cells) than a hepatic abscess usually contains, or, so far as my knowledge and experience enable me to judge, ever contains.

When, after incision and drainage, the oozing of red blood continued, the original diagnosis of a hepatic abscess, which was made before and after the first aspiration, seemed negatived. I was then at a loss to account for the nature of the condition. The most likely lesion seemed to me to be a large angioma of the liver which had broken down and formed a suppurating blood cyst. And looking back at the clinical features which the case presented during life, I still think that this was the most feasible diagnosis which could have been arrived at to account for all the facts of the case.

The notes of the case are as follows:—Mrs A. M., aged 25, was admitted to Ward 5A, Edinburgh Royal Infirmary, on 23rd June 1894, complaining of debility, emaciation, shortness of breath, and pain in the lower part of the right chest at a point  $2\frac{1}{2}$  inches below and slightly external to the right nipple.

*Previous History of the Present Illness.*—The patient states that she was quite well until her marriage, three months ago. Since then she has suffered from pain in the right side in the position described above. She thinks it was worse in wet weather. At first it was intermittent, and of a dull aching character, but lately it has been more constant, sharp, and boring. The patient attributes her illness to catching cold. At the commencement of the attack she was troubled with a dry cough, but there was no expectoration. The cough continued for two or three weeks, and then disappeared; the pain, however, remained. In other respects she has been fairly well, though getting weaker. Her appetite has been good, and her digestion good, though she has been somewhat troubled with flatulence. She has noticed that the right side of the chest has recently become more prominent than the left.

*Previous History prior to the Present Attack.*—The patient states that until the present illness commenced she always enjoyed good health. She has never lived abroad, and has never suffered from dysentery, diarrhoea, or any affection of the bowels. She last menstruated some six weeks ago.

*Family History.*—The patient is one of a family of five; four are living, strong and healthy; one died at the age of six from tubercular meningitis. Her father and mother are both alive and well. So far as she knows, none of her relatives have suffered from any liver affection.

*State on Admission.*—The patient is pale and thin; she looks extremely ill. She lies on her right side; she says that the pain is worse when she attempts to move or when she lies in any other



position. There is no jaundice. The evening temperature is about  $100^{\circ}$ ; the morning temperature  $98^{\circ}$ ; pulse varies from 110 to 120.

The right side of the thorax over the region of the liver (infra-mammary and hypochondriac regions) looks fuller than the left. The bulging is most marked in the position of the fifth, sixth, and seventh interspaces in the anterior axillary line. The sternum appears to be pushed forward, and there is some fulness in the epigastrium. A localised tender spot is present in the sixth right interspace in the anterior axillary line. The apex of the heart is displaced upwards and to the left; it corresponds to a point slightly outside and on a level with the left nipple. The liver dulness is greatly increased. In the mammary line it extends from the third rib above to a point 1 inch above the umbilicus. Posteriorly there is marked dulness over the lower part of the right chest; the dulness extends as high as the spine of the scapula; its upper border is curved, and corresponds in shape to Garland's S-shaped curved line, representing the upper limit of the dulness in a moderately large pleuritic effusion.

The breath sounds, vocal fremitus, and resonance are absent over the dull area.

On the left side of the chest the breath sounds are harsh and bronchial in character, and the vocal fremitus is well marked.

The heart appears to be normal.

The splenic dulness seems to be normal.

The tongue is red, congested, and dark-looking, not furred or tremulous. The appetite is good. The patient is troubled occasionally with flatulence. The bowels are costive.

The urine is of a dark reddish-brown colour; clear, acid in reaction; its specific gravity is 1014. The deposit, which is considerable, consists of amorphous urates and uric acid. The urea equals 1.3 per cent. The urine contains neither bile, blood, albumin, nor sugar.

*Treatment and Subsequent Progress of the Case.*—On the evening of the patient's admission to hospital a diagnostic puncture was made by my House-Physician into the right pleural sac, but no fluid was withdrawn.

On *June 25*, a hypodermic needle was passed into the right chest at the tender spot between the fifth and sixth ribs, and some dark anchovy-paste-like fluid, highly suggestive of an abscess of the liver, was removed. On microscopical examination, it was found to consist of red blood corpuscles, leucocytes, and broken-down cellular elements, apparently liver cells.

On *June 26*, 53 ounces of dark anchovy-paste-coloured fluid were withdrawn with the aspirator: the needle was passed into the liver at the position of the tender spot between the fifth and sixth ribs. The fluid which was withdrawn consisted of white blood corpuscles, red blood globules, broken-down cells, granular *débris*, and a few large cells, apparently liver cells, in process of

necrosis and fatty change. The amount of blood which the fluid contained was much larger than one expects to find in an abscess of the liver.

The removal of the fluid was followed by marked relief both to the breathing and the pain.

*June 27.*—The patient feels better to-day; she is quite free from pain; her expression is less anxious looking, but she is very pale, and the pulse is quick (120), the respirations 32, the temperature 98°·8. There was little or no rise of temperature last night. The tongue is moist, red, and speckled with a white fur.

*June 28.*—Last night she suffered a good deal from pain in the right shoulder. To-day she still complains of pain, and the cavity in the liver appears to be filling up. There is some œdema of the skin in the right hypochondriac region.

*July 1.*—The distension of the right side of the thorax is quite as marked as it was before the aspiration; the cavity in the liver has evidently filled up. The pain and difficulty in breathing are very troublesome. The patient is very pale and weak; the pulse ranges from 114 to 120; the respirations from 38 to 40. The feet are slightly swollen. Mr Cotterill saw the patient with me, and it was agreed to incise the cavity and to insert a drainage-tube.

The operation was performed on *July 2*. 50 ozs. of anchovy-paste-coloured grumous fluid, which was more bloody than that removed by aspiration, were evacuated.

*July 3.*—The patient had a very good night. She is quite free from pain, and says that she feels better. The tongue is clean and moist, the temperature 97°, the pulse 108, the respirations 36.

*July 7.*—The wound has been dressed several times; there is a considerable quantity of bloody discharge. The patient is very pale, but in other respects looks better. She says that she has been much relieved since the operation. The tongue is clean; the appetite so good that the nurse says the only difficulty is to satisfy her, for she is ravenously hungry. She is sleeping well.

*July 9.*—The bloody discharge still continues in considerable quantity, and the patient is more anæmic. In other respects she is well. To have one of Robertson's (No. 3) Bland's pill capsules three times daily.

*July 10.*—The bleeding still continues; ice-bags to be kept continuously applied to the region of the liver.

*July 17.*—The bleeding still continues notwithstanding the local application of ice and the subcutaneous injection of ergotine. The patient was so profoundly anæmic that it was decided, after consultation, to close the wound. Mr Cotterill accordingly sewed the edges of the wound closely together with horsehair, and painted collodion over the surface of the wound.

*July 18.*—The patient suffered a good deal of pain in the region of the liver during the night. There is some oozing through the

dressing to-day. Five minims of turpentine to be given every eight hours.

*July 20.*—Not a drop of fresh blood has escaped through the dressing since yesterday morning. She has only had two doses of turpentine,—one on the evening of the 18th and a second on the morning of the 19th. She feels sick, the turpentine has therefore been stopped. She complains of pain in the tip of the right shoulder. The cyst has again filled up; the right side is again much more prominent than the left; the breathing is very rapid, the patient profoundly anæmic and prostrate. With the object of relieving the pain and distress of breathing, the cyst was again aspirated, and 20 ounces of dark bloody fluid, containing some pale coagula, withdrawn. On microscopical examination the fluid consisted of leucocytes, red blood corpuscles, granular *debris*, and a few large cells, apparently liver cells.

*July 24.*—The patient feels very weak and prostrate, but is free from pain. There is a little leakage from the puncture.

*July 26.*—The breathing was so much distressed last night, that the House-Surgeon again aspirated the cyst in two places. On percussion to-day an area of tympanitic resonance is present on the right side between the fourth rib and the seventh space anteriorly.

*July 30.*—The patient died to-day.

#### PATHOLOGICAL REPORT BY DR R. F. C. LEITH.

The necropsy was made by me on the 31st July 1894, some twenty hours after death, Dr Bramwell being present.

The body was somewhat emaciated; post-mortem rigidity and lividity were slight. There was a recent linear cicatrix in the sixth right interspace. It ran parallel with the ribs for about 1 inch from the nipple line outwards. Its outer extremity showed a small aperture about the size of a crow-quill. This led by a short sinus downwards, backwards, and inwards into a large cavity. A probe could be passed into this cavity for fully 6 inches, and could be moved freely about in it.

On opening the abdomen the peritoneum was seen to be normal, but the membrane was drawn upwards and over to the left side. The liver was more prominent than usual, and projected for fully 2 inches below the costal margin in the line of the gall bladder. The left lobe extended further into the left hypochondrium than usual, and also projected further downwards. The right lobe, just below the costal margin, showed a deep groove formed by the uprising margin of a prominent smooth tumour-like swelling of a whitish, glistening, and fibrous appearance. This was seen over the whole lower margin of the right lobe, and passed upwards beneath the ribs as a large dome-like swelling. On opening the thorax and removing the sternum with the rib cartilages, the upper surface of the liver was found to be

extensively adherent by recent easily separable adhesions to the anterior part of the diaphragm. These were strongest and oldest opposite the level of the fifth rib. The large dome-like tumour already referred to was now seen to occupy the whole of the upper surface of the right lobe of the liver, and to project considerably into the left lobe, displacing the suspensory ligament fully  $\frac{3}{4}$  of an inch to the left. It was uniformly round, and had a glistening, fibrous appearance. It rose gradually upwards, reaching a considerable height above the ordinary level of the liver in the middle of the right lobe. The appearance of the groove at its lower margin was partly due to the colon, etc., pressing the thin inferior lower liver border forwards. Two small apertures were seen in the anterior surface of the tumour, a little above and to the right of its centre. These led into cavities in the tumour, apparently separate from one another, and a probe could be passed from the upper of the two through a small aperture in the middle of the diaphragm into the right pleural cavity. This perforation in the diaphragm was due to the puncture of an aspiration needle shortly before death. The right pleural cavity contained a small quantity of recent lymph. On palpation of its upper surface, the whole tumour gave a sense of semi-fluctuation more marked towards the right side than inferiorly and to the left.

On removing the liver, the inferior vena cava at its place of exit from the organ was free from clot, but at its entrance into it, and for fully 2 inches downwards in the abdomen, it contained a pale greenish lateral thrombus adherent to its right side. It was comparatively recent, not very firm or adherent, and faded gradually below into an ordinary black clot. The vein in its course through the liver was free from clot. The structures of the anterior layer of the gastro-hepatic omentum, the hepatic artery, bile ducts, and portal vein were normal and in no way obstructed. The portal vein contained no clot even in its main lateral branches. The liver weighed 9 lbs. 6 ozs., and measured  $11\frac{1}{2}$  inches transversely, 10 inches antero-posteriorly, and fully  $4\frac{1}{2}$  inches in thickness. The tumour was now seen more fully, and occupied, as already mentioned, nearly the whole of the right lobe and a part of the left. It projected above the upper surface of the organ, though it hardly reached its under surface, which presented practically a normal appearance. It measured 7 inches in transverse diameter,  $7\frac{1}{2}$  inches in antero-posterior diameter, and about 4 inches in thickness. The gall bladder was small and contracted, and did not show below the lower liver border. It contained a small quantity of olive-green viscid bile.

An antero-posterior section was made through the middle of the right lobe. It showed the tumour mass occupying the whole of the right lobe, replacing the proper liver tissue everywhere except for 1 to  $1\frac{1}{2}$  inches towards the inferior border, and a mere

shell on part of the under surface. It was everywhere encapsulated by a firm fibrous-looking capsule of varying thickness.

The main mass of the tumour was composed of a structureless, red, granular, and friable material, obviously crumbling blood-clot. The rest showed lobed and irregularly rounded masses of a soft whitish-yellow gelatinous-looking substance, reminding one somewhat of a gelatinous blood-clot. These were situated towards the inferior and posterior part of the liver. They were seen, on the one hand, to be clearly defined and bounded by the limiting external capsule, and, on the other hand, to irregularly mingle with the hæmorrhagic mass. The largest area was about the size of an ordinary orange, oval in shape, with a clearly defined fibrous-looking wall around it, separating it from the hæmorrhagic mass. The same capsular appearance was seen elsewhere, but not so distinctly; and in all cases the contained gelatinous material could be easily detached, and suggested the idea of a multilocular cyst into which the jelly-like substance had grown as solid ingrowths from part of the wall. Other antero-posterior sections were made both to the right and left of this section, and showed irregular and rounded areas of the gelatinous substance mixed up with red areas, evidently consisting of pure blood-clot or of the gelatinous material infiltrated more or less intimately with blood. What appeared to be white fibrous septa penetrated the whole tumour in several directions. Everywhere towards the left the encapsulation seemed to be perfect, but near the right border there were a few places in which it was scarcely detectable. In fact this part showed an appearance as if separate roundish centres had become confluent. A comparison of the appearances presented by all these sections led me to believe that the more actively growing part of the tumour lay towards the right and the oldest part to the left.<sup>1</sup> The liver substance, where it was still visible outside the tumour, showed a distinct fatty change. It was paler than normal, and presented irregular areas of congestion, but nothing else abnormal. The branches of the portal vein throughout were free from clot, and the other structures of the portal spaces presented normal appearances. None of the bile ducts were dilated. Some of the sublobular veins showed small recent thrombi in the

<sup>1</sup> It is a somewhat remarkable fact that in no part of the tumour could I find any trace of such a cyst as could have held the amount of chocolate-coloured fluid which was drawn off during life. In fact, so far as the naked eye could judge, there were apparently no obvious cysts present. A large number of cysts of various sizes was, however, found microscopically to be present. On passing the probe through the aperture in the capsule already mentioned, it was seen to enter into the large hæmorrhagic mass. It is, therefore, probable, that this was the source of the fluid, and that during life it had been a cystic mass into which hæmorrhage gradually took place, and the clotting may have been largely just ante-mortem or partly even post-mortem.

neighbourhood of the clot, evidently secondary in their nature.

No growth of any kind was found anywhere else, either in any of the thoracic or abdominal viscera.

The spleen weighed 5 ozs. It was firm and pale, but healthy.

The left kidney weighed 4 ozs., and the right  $4\frac{1}{2}$  ozs. They were both small, and paler than usual; but beyond a slight waxy degeneration affecting some of the Malpighian bodies, they appeared to be healthy.

The stomach and intestines were quite healthy, as were also the mesenteric and retro-peritoneal glands. The bladder, vagina, uterus, ovaries, etc., were all carefully examined and found to be normal.

The heart weighed 7 ozs. Its valves were competent and healthy. The myocardium was somewhat atrophied and pale.

The left lung weighed 14 ozs. and the right only 10 ozs. The upper lobe in both was anæmic, and the lower œdematous. There was some collapse on both sides, but especially on the right, towards the base and root. There was also some recent lymph on the base of the right lung and on the corresponding surface of the diaphragm around the puncture.

The brain weighed 48 ozs. It was healthy, and no other lesion of any kind was found in any other structure in the body.

*Microscopic Examination.*—Sections of the margin of the tumour showed a well-defined fibrous capsule separating it from the condensed and somewhat cirrhotic liver tissue. The structure of this marginal part of the tumour was mainly cellular, consisting of spindle and irregularly-shaped cells in a reticular-looking matrix of finely fibrillar connective tissue. The cells varied much in size, and resembled sarcomatous cells of a somewhat aberrant type. Some of them were very large, with convoluted or multiple nuclei. Such giant cells, however, are not infrequently found in spindle-celled sarcomata. Many persistent bile ducts were to be seen in this part of the section. Sections of any of the gelatinous areas showed a structure characteristic of a medium-sized spindle-celled sarcoma. The cells were long and spindle-shaped, the nuclei oval and almost rod-shaped in places. Indeed, here and there the cells closely resembled non-striped muscle cells. The intercellular substance was scanty and the bloodvessels rudimentary. Sections of the material in the large blood cyst or hæmorrhagic-looking area proved to be ordinary blood-clot. Towards its margins it became mixed with tumour cells. Many small branched cysts lined with a single layer of endothelium were seen both in the typical sarcomatous part and at the margin of the hæmorrhagic area. They were free from contents, except here and there where the hæmorrhage around them had passed into them and partly filled them. Microscopic hæmorrhages were also plentifully seen in nearly every part of the tumour.

*Nature of the Tumour.*—It was undoubtedly a sarcoma, tending towards the spindle-celled variety. On account of its numerous bloodvessels, and the multitude and magnitude of its hæmorrhages, it might be called an angeio-sarcoma. The cysts were hardly numerous or important enough to entitle it to be called a cystic sarcoma.

*Origin of the Tumour.*—After sections of many parts of the periphery were made, some were found showing an early stage of the growth. They showed columns and islands of liver tissue surrounded by the tumour cells. The latter were seen pushing themselves outwards between the columns of liver cells, and as the endothelial cells of the bloodvessels could sometimes here be made out, the tumour cells apparently occupied the place of the perivascular lymphatics. This naturally suggested an origin in the connective tissue cells lining these lymphatics. If any safe deduction can be made as to the origin of any sarcoma from the behaviour of its growing margin, then there is clear histological evidence in this case of the tumour having originated in the connective tissue cells lining the perivascular lymphatics of the intralobular capillaries of the liver. Block believes that the origin in his case was from the endothelium of these capillaries; while Hörup, in his, traces it from the adventitia of the hepatic arteries.

The literature of primary liver sarcoma is not of a very satisfactory kind. I have found a record of about 33 cases, few of which, however, can be regarded as true examples. Of these, six were melanotic and three lympho-sarcomas, more or less diffused. All except three of them presented the appearance of many nodules scattered more or less widely throughout the liver substance. In these three there was a large mass in the right lobe, partly gelatinous, and partly mixed and infiltrated with blood. One was in a child 8 months old, recorded by West. He speaks of it as a cancer, and says there were secondary nodules in the lung. From its description I think it may be justly regarded as a sarcoma. The second case is recorded by Roberts, under the title of "A Case of Fungus Hæmatodes of the Liver." It occurred in a girl of 12, and formed a large mass weighing about 8 lbs. in the right lobe. The third case is recorded by Hörup of Copenhagen. It occurred in a woman of 26, and formed a mass in the right lobe measuring about 5 inches in all directions. It was a small round-celled sarcoma.

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#### BIBLIOGRAPHY.

1. VIRCHOW.—*Die Krankh. Geschwülste*, Bd. ii., s. 276. Melanotic.
2. ACKERMANN.—*Die Histogenese und Histologie der Sarkome*, s. 32. Melanotic.
3. FRERICHs.—*Klinik der Leberkrankheiten*, Bd. ii., s. 321, 1861. Melanotic.

4. NAUNYN.—“Ueber eine eigentümliche Geschwulstform der Leber,” in *Archiv für Anat. u. Phys.*, von Reichert u. Du Bois Raymond, s. 710, 1866. A cysto-sarcoma.
5. HÖRUP.—*Hospitalstidende*, 10 Jahrb., No. 1; *Jahresbericht* von Virchow und Hirsch, Bd. i., s. 284, 1867. A small round-celled sarcoma.
6. MILLARD.—“Diathese sarcomatense. Baste sarcome du foie,” etc., *L'Union médicale*, Nos. 122, 123. Spindle-celled sarcomata (doubtful cases).
7. E. WAGNER.—“Die heterologe geschwulstformige Neubildung von adenoider oder cytogsnar Substanz,” etc., *Archiv der Heilkunde*, s. 45, 1865. A lympho-sarcoma.
8. PELLACANI.—“Sarcoma fuscicolato del fagato,” etc., *Rivista clinica di Bologna*, 1880; Virchow u. Hirsch's *Jahresbericht*, Bd. i., s. 271, 1880.
9. ROBERTS.—“A Case of Fungus Hæmatodes of the Liver,” *Lancet*, i. p. 77, 1867.
10. PARKER.—“Diffuse Sarcoma of Liver, probably Congenital,” *Trans. Path. Soc.*, vol. xxxi. p. 290, 1880.
11. LEDUC.—“Sarcome mélanique du foie,” *Progrès méd.*, viii. 591-593. Paris, 1880. (Secondary to melanotic sarcoma of eye.)
12. MEISENBACH.—“Myxosarcoma of the Liver in an Infant Four Months Old,” *Weekly M. Red.*, ix. 433. St Louis, 1884.
13. BURNET.—“Primary Melanotic Sarcoma of Liver” (card specimen), *Trans. Path. Soc.*, vol. xxxvi. p. 252, 1885.
14. TOOTH.—“Diffused Lympho-Sarcoma of Liver,” *Trans. Path. Soc.*, vol. xxxvi. p. 236, 1885.
15. WINDRATH.—“Ueber Sarkombildungen der Leber,” I. D. Freiburg, B., p. 20, 1885.
16. WEST.—*Diseases of Children*, p. 764, 1884.
17. HUTYRA.—“Adatok a majdaganatok tanaloz” (Sarcoma of the Liver), *Orvosi hetilap*, xxx. p. 409, 1886.
18. REHN.—*Tageblatt der 60 Versammlung deutscher Naturforscher und derzte in Wiesbaden*, s. 313, 1887.
19. PODROUZEK.—*Prager med. Wochenschr.*, No. 33, p. 351, 1888.
20. BLOCK.—“Ueber ein primär melanotische endothelium der Leber,” in *Archiv der Heilkunde*, Leipzig, s. 412, 1875.
21. ORTH (M.).—“Ueber primäres Lebersarkom,” I. D. Strassburg, 1885.
22. ARNOLD (J.).—“Zwei Fälle von primärem Angiosarkom der Leber,” *Ziegler's Beiträge*, Bd. viii. p. 123, 1890.  
Etc., etc.

Dr Affleck said that this paper recalled very vividly to his mind a case that occurred in the Royal Infirmary a few years ago, which might interest them. In the first place he must refer to the difficulty of diagnosis in these cases. The more one saw the less one was likely to dogmatise. The case occurred in this way:



An Italian woman, a street musician, was treated in the Fever Hospital for typhoid fever. She had all the symptoms of typhoid, and was in the hospital for three months. A swelling was observed in the right side. She was admitted to the Infirmary, and he (Dr Affleck) found a large fluctuating tumour under the right ribs, and to all appearance in the liver. On a puncture being made, chocolate-coloured matter came out. Mr Duncan saw the case with him. The woman was very ill, and had a high temperature. She had very little acquaintance with English, so that they could not obtain a history. Surgical interference was decided on. He thought it might be a suppurating hydatid cyst. Mr Duncan cut into a huge cavity in the liver. A large quantity of grumous matter came away, and Mr Duncan introduced a drainage-tube. The woman lived only a short time, and after death there was found an enormous abscess cavity in the liver, communicating with a gastric ulcer, which opened by a sinus into the cavity, and in and out of which the contents of the stomach passed. Such things, they knew, happened over and over again, and that was one of the points to remember in connexion with these fluid tumours of the liver.

### 3. SHORT NOTES OF TWO CASES OF OPIUM POISONING.

By JAMES RITCHIE, M.D., M.R.C.S. Eng., F.R.C.S. Ed., F.R.C.P. Ed.

THE following two cases of opium poisoning seem to possess features of sufficient interest to warrant the presenting short notes of them to the Medico-Chirurgical Society.

CASE I.—About 6.30 on the morning of 12th April 1895 I was hurriedly summoned to a case, said to be one of poisoning. I took with me a syphon apparatus for washing out the stomach, and on arriving at the house was informed that the patient had dined heartily the previous evening about seven o'clock, that some hours later he had experienced severe pain over the heart, for which he had taken a dose of laudanum, and the friends were afraid that he had taken an overdose. The patient, a gentleman about 40 years of age, was in bed, pale, breathing slowly, pin-point pupils; he could be roused with difficulty, answered in monosyllables, and if left alone relapsed at once into somnolence. I at once injected subcutaneously about  $\frac{1}{60}$  grain of atropine sulphate, got him out of bed, and with some trouble kept him awake in a chair until the cesophageal tube was passed and the stomach was washed out. A large quantity of partially digested food was removed with some difficulty, because of the occasional blocking of the tube. The material smelt strongly of opium, and it contained a large amount of mucus. The stomach was washed out till the water returned almost clear, then strong coffee was given, and assistants were obtained to keep the patient walking about. As the pupils were still small, a second subcutaneous injection of atropine was given,

on this occasion  $\frac{1}{150}$  grain. In the forenoon the patient was still drowsy, and craved to be allowed to sleep. As the day advanced he was permitted to rest at intervals. In the evening he was out of danger, and allowed to go to bed. It was found that the dose of laudanum taken was 2 ozs. Although he had occasionally taken small doses for sleeplessness, he was not in the habit of using opiates. The chief point of interest in this case is that although a large dose of a fluid preparation of opium was taken at night, it was found that six or seven hours afterwards the stomach still contained a large quantity, and therefore the symptoms of opium poisoning were not so profound as might have been expected in view of the long time which had elapsed after the taking of such a dose. The explanation is doubtless—*First*, that the dose had been taken when the stomach was full of food; *second*, that in consequence of well-marked gastric catarrh the dinner was only partially digested, and for the same reason only a small portion of the laudanum had been absorbed. This is a demonstration on a somewhat large scale of facts with which we are conversant in practice, viz., that for the immediate relief of severe pain in a patient who has much in the stomach, or in one who is the subject of severe gastric catarrh, it is of little avail to give morphine by the mouth. In such cases it should be given hypodermically.

CASE II.—On July 24, 1895, I was called between one and two o'clock A.M. to see a somewhat small man who had taken, not very long previously, about an ounce and a half of laudanum. He presented the ordinary symptoms of opium poisoning. The pupils were very small. He could be roused without much difficulty, but could not be made to give any account of his previous proceedings, and when left alone he at once fell asleep. Having shortly before that time read in the *British Medical Journal* a paper by Dr Moor, of New York, on "Permanganate of Potash as an Antidote in Opium Poisoning," I gave, in a large cupful of water, about an ounce of Condyl's fluid, which was fortunately at hand in the patient's house, then went to get my syphon apparatus. Immediately after returning, the patient's stomach was washed out until water, with a small quantity of Condyl's fluid added to it, returned unchanged in colour. A little Condyl in water was then given to drink, and the patient was kept moving. He remained drowsy until the forenoon. This case seems to afford a good example of the power of potash permanganate to render innocuous preparations containing morphine.

Dr Moor (*British Medical Journal*, June 1895, p. 1369), after having experimented with morphine preparations, convinced himself that the morphine is oxidized, and that the manganese salt is reduced to hydrated manganese dioxide. He found that sulphate of morphine is decomposed much more rapidly than

albuminous matter, such as white of egg,—that one grain of permanganate oxidizes one grain of morphine. Such confidence had he in the power of this remedy that, although he is very susceptible to the action of narcotics, he took on one occasion 3 grains of sulphate of morphine, followed in thirty seconds by 4 grains of potassium permanganate; and on another occasion, two hours after breakfast, he took 5 grains of sulphate of morphine, followed in a few seconds by 8 grains of the antidote dissolved in 8 ounces of water, and in neither case did he experience any effect from the narcotic. Recently in the *British Medical Journal* (May 16, 1896, p. 1193), Dr Luff, of St Mary's Hospital, records a series of experiments in which he mixed a known quantity of morphine acetate with vomit, and after thorough agitation a known quantity of potassium permanganate in solution was added. After a time careful examination was made, but not the faintest trace of morphine could be discovered. These experiments demonstrated that this salt of morphine is more readily oxidized than other stomach contents. Dr Luff advises that if laudanum has been taken and the quantity is known, 6 grains of permanganate should be given for every ounce of laudanum, that the stomach should be washed out, and as morphine is partly excreted into the stomach, it should be washed out two or three times at intervals of half an hour. As the antidote seems to be so certain in its action, such an unpleasant proceeding is surely unnecessary. Is it not sufficient after the first washing to give small doses to be swallowed?

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*Dr F. D. Boyd* said that he had much pleasure in rising to thank Dr Ritchie for his very interesting paper. There was only one point in reference to it that he would emphasize, viz., the purely local and chemical action of the permanganate in the stomach. An interesting case occurred the other day where a very young enthusiastic medical man gave hypodermics of permanganate, with the natural effect of producing local suppuration. It would be well to insist on the point, that permanganate was to be given only by the mouth, and not hypodermically.

*Dr H. M. Church* said that Dr Ritchie had done well in bringing before them this very simple and evidently very effectual way of treating opium poisoning with permanganate of potash. He should like to point out that it was not uncommon to give nursing mothers a little opium for after-pains. Dr Ritchie had just asked him if he had had any bad effects on the infant. He had not had any such effects in giving opium to mothers, although the child was put to the breast very shortly afterwards. He thought it was an interesting point in connexion with this discussion. It was still to be explained whether the oxidation of it took place before it reached the child or not. At all events, it was the case that it did not seem to affect the child.

## Meeting XI.—July 1, 1896.

Dr WILLIAM CRAIG, *Vice-President, in the Chair.*

## I. EXHIBITION OF PATIENTS.

1. *Dr Allan Jamieson* showed a case of ACANTHOMA. He had never seen a similar case, nor did he know of any illustration of a skin disease resembling it. A. E., 20, a native of Perth, admitted to Ward 38 on May 30, 1896. Family history good. She has always been rather delicate, is stunted in growth, and has never menstruated. For the last seven years has suffered from bronchitis in winter, and has still a cough. Her complexion is sallow, and she is thin. Hair has a dry and faded appearance, sandy in colour. Digestive and circulatory systems normal. Urine contains no abnormal constituents. Her skin disease commenced when she was six weeks old on arms, and thence spread over body. Though treated in various institutions with some benefit for the time, she has never been wholly free from the complaint. It has been much worse since she was fourteen. When admitted to the Royal Infirmary the cheeks, forehead, and upper lip had pinkish-coloured infiltrated and crusted oozing patches, indistinguishable from a moist eczema. Scattered over the back, scapular regions, at root of neck, down the flanks, but less over the spine, and on the arms and legs, were rose-pink patches not very distinctly margined, and feeling a little thickened on pinching. These were dry, scaly, and rough. Between these were numerous blotches of a pinkish hue, the skin itself being sallow. On the front of the trunk there were similar patches, which over the clavicles were arranged in lateral lines, on the hypochondriac region in dots. On the inner side of the thighs, extending to and below the knee, and on the areola of the mammæ, the condition reached its acme. It there looked quite like mucous tubercles, covered with brownish crusts. The patches were elevated from two to three lines, were uneven, nodulated, or mamillated, blunt papillary elevations. The elevations were firm though soft to feel, and their margins were sharply defined against the healthy skin, from which, indeed, they rose abruptly. The eruption bore evidence of scratching, and was itchy. The inguinal glands were enlarged. Boric starch poultices and boric acid baths continued for a few days removed all crusts and rendered the surface clean and smooth. From this time onwards the sole treatment consisted in inunction with a ten per cent. salicylic vaseline. By the end of June the greater part of the eruption was represented by smooth, flat, pinkish-purple patches, little if at all thickened, and now the source of no inconvenience. She had put on flesh, and looked plump and well. But the elevations on the inner aspect of the thighs and areolæ had not flattened down. They still presented smooth, closely-packed nodular prominences or projections. Most

of these were a pale purplish-pink, with here and there a few of a darker brown tint. On pressure the colour faded entirely, and only returned after a few seconds, and then slowly. The marked thickening was perceptible on pinching up. One entire nodule, a quarter of an inch across, was cut out soon after her admission, and was prepared for examination by Dr Leith. The horny layer of the epidermis was thin but continuous. Its thinning may have been partially due to the salicylic acid, which had been used for a short time. There were long finger-like, sometimes branched, prolongations of the epidermis running down into the corium; these were composed mainly of well-formed prickle cells. The intercellular lymph spaces were dilated, and the cells themselves oedematous. The meshwork of the papillary corium was opened out, the bloodvessels dilated. The deeper layer of the corium showed some degree of cellular infiltration. Beneath this there was much firm connective tissue, arranged in wavy bands. Round the coil glands there was a myxomatous tissue, with branching connective tissue cells. The glands themselves were little altered. The condition, therefore, is an acanthoma, with secondary oedema. It approximates in structure, on the one hand, to eczema in an exaggerated degree; on the other, to the structure of a condyloma, and to the condylomatous-like patches found in the rare disease called acanthosis nigricans of Pollitzer, Janovsky, and Malcolm Morris. But for the present the case is unique.

2. *Dr G. A. Gibson* and *Mr Alexis Thomson* showed a patient after EXCISION OF PYLORUS FOR CANCER, also naked-eye and microscopic specimens of two cases of CANCER OF THE PYLORUS.

*Dr Gibson* said that the patient, a woman aged 46, was sent across to him by *Dr Goodenough* of Dysart in the month of April. She had presented every symptom of dyspepsia for five or six months. She was wasting rapidly, and there was in addition, on examination of stomach contents, total absence of free hydrochloric acid, and presence of lactic acid, with a very much distended stomach. There was underneath the origin of the right rectus considerable resistance on palpation. From all these symptoms he came to the conclusion that there was probably pyloric cancer. The patient was in the Deaconess Hospital. He asked his colleague, *Mr Alexis Thomson*, to see her with him. They concluded to perform laparotomy to remove the cancerous growth of the pylorus, as it seemed an eminently suitable case. The pyloric cancer was removed, and since that time the patient had made an uninterrupted recovery. When she first came under observation she was 6 st. 1 lb. in weight. After operation she was fed on peptonized food in small quantities by the rectum, and rapidly lost weight, but she was now 6 st. 10 lbs. in weight. She could now digest well, could walk a good many miles, and was in perfect health.

*Mr Alexis Thomson* said that, as neither of the patients had

arrived yet, he would show the two specimens. On opening the abdomen they found a very localized cancer in the region of the pylorus, and two glands in the greater curvature immediately below the pylorus. In several respects the case was eminently suitable for removal,—a very circumscribed cancer, and only two glands. They proceeded to removal after the method recommended by Kocher. To those familiar with the system of Kocher he need not recapitulate the steps. Suffice it to say that they depended very largely on clamps for prevention of any escape of gastro-intestinal contents while isolating and removing the cancerous segment of the intestinal canal. The specimen removed was shown. He might say that, on account of the hurry they were in, they plunged it straight in spirit, and it made a most tremendous difference whether a tumour like this was put straight in spirit, or, as it should be done, placed for twenty-four hours in running water. The other pylorus now shown was placed in water for twenty-four hours, so that the muscle was completely dead, and no longer contracted in the spirit. But the specimen placed in a vital condition in the spirit contracted in an extraordinary way. What was a fair margin of healthy tissue round the cancer now appeared as if in dangerous proximity to the cancer. Having excised the tumour, they cut into the stomach; then, by means of Murphy's button, they united the posterior wall of stomach and duodenum. Although the operation was done on the 9th of May,—*i.e.*, forty-seven days ago,—the Murphy's button, which acted so well, had not yet been passed. Dr Dawson Turner had been trying to identify its position by skiagraphy, and although they had not been able to obtain photographs, they saw by the efflorescent screen that it was somewhere between the umbilicus and the ensiform cartilage,—*i.e.*, somewhere in the position in which it was originally placed in the patient. They did not despair of seeing this button again. Probably it would appear in the course of the next few months. They should certainly be more at ease when the button had been found. The patient was shown. It was a little difficult, Mr Thomson said, to realize now her condition before operation. She was then very much emaciated and helpless. Dr Gibson had already mentioned the great addition to her weight. The wound healed by first intention, and the abdomen was now firm and natural.

*Dr Gibson* added that he did not think that surgeons had been allowed a very fair chance in this matter, because most of them on the medical side had been inclined to wait until they could feel a tumour sometimes as big as their heads. It was advisable that, when they could infer from the symptoms that pyloric cancer was probably present, they should at once proceed to operation.

*The Chairman* said he thought the Society must congratulate Dr Gibson and Mr Alexis Thomson on the successful diagnosis and treatment of this very interesting case.

3. *Mr Alexis Thomson* then showed a patient after REMOVAL OF SECOND DIVISION OF FIFTH NERVE (by preliminary resection of malar bone) for epileptiform neuralgia. This was, he said, quite a characteristic case of severe trigeminal neuralgia. For fully two years this man's life had been made miserable by really very severe epileptiform neuralgia coming on in spasms, which absolutely incapacitated him from doing anything, occurring at frequent intervals, sometimes three or four in the day, and were associated with convulsions of the facial muscles,—regular convulsive type of facial neuralgia. As the neuralgia was mainly confined to the distribution of the second division of the fifth, they determined to remove that very thoroughly. They had attempted to do this before by what was known as Carnochan's process, and were not satisfied that they got away the entire nerve. They determined in this instance, in consultation with his friend Mr Harold Stiles, to follow out the method described very fully in Kocher's text-book, viz., to expose this nerve by reflecting the malar bone. The malar bone was detached from its surroundings on all its aspects save the posterior. They turned it right out in this position, so that they could trace up the superior maxillary division of the fifth right back to the foramen rotundum. They divided it, and removed it from the face as far as desired. The patient was for the present completely relieved, but it was done only a few months ago, and they did not know if it would be permanent. There was no anæsthesia in the area of distribution of the nerve. He had still tactile sensibility. He (Mr Thomson) was a little disappointed with the appearance of this patient. The wound healed straight off, and on removing the collodion the appearance was very good,—not any more than one of the natural lines on the face; but since the patient had been in the country, evidently from consolidation of malar bone, the scar had become depressed, and there was distinct deformity, more than they expected, without, however, any drawing down of the eyelid.

4. *Dr Harry Rainy* showed a case of PARALYSIS AGITANS with some unusual symptoms. The disease, he remarked, began at a very early age, viz., thirty-three years, and the patient had now suffered from it for about thirteen years. His work exposed him to toxic vapours for some time before the disease came on; but the immediate cause seemed to have been a fall from a ladder, 6 feet high. There was no obvious injury at the time, but he felt a good deal shaken, and continued to have pain in the dorso-lumbar region. About six months later tremor began. It advanced in the typical way,—arms first affected, afterwards legs. He now exhibited all the characteristic symptoms,—the tremor, the festinant gait, difficulty in turning, retropulsion, and sluggish rotation of the head. He had great difficulty in maintaining his balance in rising. As a rule, the tremor was increased very markedly by any activity

on his part. Generally when he was sitting in the consulting-room his tremor entirely ceased unless something occurred to agitate him. Once he had performed a movement he could go on repeating it with comparative ease, and the exercise temporarily steadied him. For example, after having repeatedly swung his arms about, he could for a short time hold up his hand quite steadily; then tremor gradually set in, at first fine, but ultimately attaining a very considerable amplitude. Movements were more readily performed against opposition. In feeding himself he had great difficulty in using a teaspoon, with a full cup he could manage more easily, whilst he could lift a heavy beaker with a pound of pellets in it to his mouth without the least unsteadiness. Besides the typical slurring speech, he had several special difficulties in articulation. These were for the most part connected with labial consonants and with explosive letters. There was also another symptom apparently peculiar to this case. From time to time a little tremor started in the eyelids and after it had gone on for some time the eyes shut entirely, and he was powerless to open them again till he raised the lids with his hand. If he raised the left lid with his hand the right eye would open of itself, but when he raised the right eyelid no effect was produced on the left eye, which continued closed. The levator palpebræ superioris was evidently affected. So far as could be ascertained, the eye symptom noted has never been recorded in any case of this malady.

### III. EXHIBITION OF INSTRUMENTS.

*Mr C. W. Cathcart* exhibited O'DWYER'S & COLIN'S (PARIS) PATTERNS OF INTUBATION INSTRUMENTS. He said he thought the members of the Society would be interested in seeing these two instruments. In the first place he did not think intubation had been sufficiently recognised and practised in Edinburgh; and in the second place, seeing the terrible nature of diphtheria, he thought anything in the way of improvement ought to be published and known, and he thought intubation specially likely to be increasingly used since the introduction of antitoxin. In order to get the best instrument he got Mr Young to write to America and ask Dr O'Dwyer to send the instrument he recommended, and he had received this (details explained). Afterwards, however, a friend of his who had been visiting the Hôpital pour Maladies des Enfants in Paris, so strongly recommended Colin's modification of O'Dwyer's tubes, that he (Mr Cathcart) got over this instrument, and was much struck with its greater simplicity. O'Dwyer's introducer and extractor had each three or four screws, but Colin's could be detached in two seconds without undoing any screws at all, and could be quickly and easily cleaned. The method of attaching the tube-rod to the introducer was also very much improved. There was no screw. It was done simply by a lever and in a moment it was attached. It could very easily be taken



to pieces, and after an operation for diphtheria could be thoroughly disinfected. The next case might be one of œdema of the glottis extending to larynx, and it might be of the utmost importance to introduce no organisms into the inflamed larynx. O'Dwyer's introducer was not easily cleaned. There was a tube which could not be dried without undoing some screws. This was a distinctly troublesome thing, as he himself had been made painfully aware. After using the instrument he boiled it, cleaned it, dried it, and put it away, and did not require it for several weeks. In the next case, while driving rapidly along to intubate, he thought he would make sure that the instrument was working. He tried to push, and found it was caught so that he could not move it. There must have been a drop of water in the fitting, causing rust, which jammed it. With the help of a cutler, after half an hour's work, he got the rust off, got it oiled, and got it to work. Of course that would never occur with him again, but it entailed extra trouble, and was a risk to which the modification was not liable. The case of O'Dwyer's was leather, to which dirt might cling. The other was metal, which could be thoroughly cleansed. Colin's had a shorter tube. The method of extraction used by him in the Paris hospitals was a beautifully simple one. He did not extract. He simply put his thumb on the child's larynx and pushed the tube out, *i.e.*, "expressed."

#### IV. ORIGINAL COMMUNICATIONS.

##### 1. ON CONTRACTION OF THE ARTERIES OF A LIMB THE PROBABLE CAUSE OF MUSCLE ATROPHY IN TUBERCULAR JOINT DISEASE.

By A. G. MILLER, M.D., F.R.C.S. Ed., Lecturer on Clinical Surgery ;  
Surgeon to the Royal Infirmary, Edinburgh.

MR PRESIDENT AND GENTLEMEN,—My object this evening will be to show that in all probability the muscle atrophy which accompanies tubercular disease of a joint is due to diminished blood-supply caused by contraction of the main bloodvessels of the limb.

Two years ago, in two papers<sup>1</sup> read before this Society, I mentioned that I had frequently noticed, when amputating for tubercular joint disease, that the main bloodvessels were unusually small, and I associated this with the atrophy which is so characteristic of tubercle. I was all the more inclined to take this view of the matter because I have been unable to find any satisfactory explanation of muscle atrophy. Hilton<sup>2</sup> has pointed out that the

<sup>1</sup> "On Bier's New Method of Treating Strumous Disease," *Edinburgh Medical Journal*, February 1894. "On the Diagnosis of Tubercular Joint Disease," *Edinburgh Medical Journal*, November 1894.

<sup>2</sup> *Rest and Pain*, p. 150.

same nerves that supply a joint, supply also the muscles acting on that joint, and the skin over the insertion of these muscles. This explains the fixation and flexion of diseased joints, but not the muscle atrophy. Now, it seems to me that diminished blood-supply from a contracted main vessel provides an explanation.

To guard against misapprehension in the course of my argument which follows, I wish to say that I am dealing with the atrophy connected with tubercular joint disease solely, not because I suppose it is the only or most marked form of muscle atrophy, but because I have studied it specially, and because the subject of muscle atrophy in a general sense would require more time and knowledge than I have to give to the subject. I do not assume that I am able to prove my argument. All I claim is that I am submitting a likely explanation of the phenomena which I have observed.

There is but one matter of original observation in my paper, viz., the association of contracted vessels with tubercular joint disease. This, so far as I know, has not been described before I drew attention to it two years ago. I regret that I have not been able to work out this from the experimental point of view, having no facilities.

In order to shorten this paper, I shall take as granted that the muscle atrophy which we find so marked in tubercular joint disease, is greater than can be accounted for by mere want of use (though my friend Mr Cathcart would not subscribe to this), and that the atrophy is caused by a reflex influence acting through the anterior horns of the spinal cord. In regard to the exact way in which this reflex acts there seems, however, to be a difference of opinion among physiologists and pathologists. Some believe that there are special trophic nerves supplying the muscles along with the motor nerves. Others consider that the vaso-motor nerves are the true trophic nerves. I will not, however, enter upon a discussion of this subject. I do not consider myself qualified to do so, and I do not at present think it necessary to establish proof one way or another. I will merely say that to my mind it seems highly probable that vaso-motor influences govern muscle nutrition, *vaso-dilatation* causing increase of growth, and *vaso-contraction* causing atrophy. I employ these terms in a general sense, viz., increase or diminution of vascular calibre from any cause.

Supposing, therefore, for the sake of my argument, that the vaso-motor system controls muscle nutrition, I suggest that probably the cause of muscle atrophy in tubercular joint disease is *a diminution of the blood-supply from contraction of the main artery or arterics*. In support of this I would point to the following facts:—

1. Contraction of the arteries exists in cases of tubercular joint disease. This I have frequently seen, and I show you to-night a leg recently amputated which exhibits the condition very markedly.

In my paper on Bier's method of treating tubercular joint disease by congestion, referred to above, I use the following expressions:—"There is a condition associated with tubercular joint and bone disease which I have observed, but which, so far as I know, has not received much attention, viz., that the arteries supplying the limb are unusually small." In my paper on the Diagnosis of Tubercular Joint Disease I say also,—“The atrophy is of the limb as a whole, muscle and bone alike, and it tends to persist. What is the explanation? I believe that the cause is a reflex contraction of the arteries of the limb, which interferes with nutrition. I have been led to that opinion by observing, when I have had occasion to amputate for tubercular joint disease, that, though there may be many active vessels in the neighbourhood of the disease, the main arteries are always very small. I have often observed the latter about half the size one would expect them to be under ordinary circumstances,—in a primary amputation, for instance.” I conclude, therefore, that contraction of the main bloodvessels exists in tubercular joint disease.

2. This brings me to my second point, which is the co-existence of arterial contraction and muscle atrophy, not only in cases of tubercular joint disease, but also under other circumstances. For instance, it is proved by Thoma<sup>1</sup> to occur in stumps after amputation. I shall refer to this again. In the meantime I turn to the question,—If these two co-exist (as they certainly do), which is the cause and which the effect, or are they both the result of a common cause?

It seems to me that muscle atrophy is not likely to be the cause of diminished blood-supply. It is much more likely to be the result. They are both symptoms of the same disease, viz., tubercular joint disease, but they may be so because the one depends upon the other, and, as I have already said, diminished blood-supply is more likely to be the cause than the result of muscle atrophy.

In speaking of atrophy generally, Thoma<sup>2</sup> gives three causes or explanations: 1, Want of use; 2, vaso-motor disturbance; 3, possible trophic nerve influence. All of these influences may, I think, imply diminished blood-supply. For instance, “want of use” means lessened demand for blood; vaso-motor disturbance that produces atrophy is likely to be of the nature of vaso-contraction, and we have seen that the true trophic nerves may be the vaso-motor system. I think, therefore, that arterial contraction, and consequently diminished blood-supply, is a cause, and a very likely one also, of atrophy. I have recently come across statements which tend to support this view. Mr W. G. Spencer, in his Erasmus Wilson Lectures on the Pathology of

<sup>1</sup> *Text-book of General Pathology*, by Richard Thoma, translated by Alexander Bruce, M.A., M.D., etc., 1896 (p. 301).

<sup>2</sup> *Loc. cit.*, p. 446.

Bone,<sup>1</sup> says, "A diminution in the arterial blood-supply checks the formation of bone whilst absorption continues, so that eccentric atrophy is brought about." And again he says, "Atrophy from disuse, and consequently diminution in the blood-supply, suffices to explain the changes in bone without the need of supposing a loss of an efferent trophic influence."

3. My next point is that the muscle atrophy which is found in connexion with tubercular joint disease is simple. This I have referred to in my former papers, but may here quote two extracts from Charcot<sup>2</sup>—"In joint disease the muscle atrophy is simple."<sup>3</sup> "There is no reaction of degeneration."<sup>4</sup> Indeed, he dwells strongly and frequently on the muscle atrophy from joint disease differing from that due to nervous causes or want of use. Others have specially examined the muscles atrophied in tubercular disease, and have found no degeneration, fatty or fibroid. I have had the reaction of degeneration tested for in tubercular joint muscle atrophy, and have found it absent. Now, this is just the kind of atrophy that we would expect to find as the result of deficient blood-supply from contracted bloodvessels. Before leaving this point I must say that muscle degeneration is found in far-advanced and old-standing cases of tubercular joint disease, in which the whole limb is atrophied. But this is only what one would expect, seeing the nerves probably share in the atrophic changes after a while, and the functions of all the various parts and tissues are more or less interfered with.

4. My fourth point is that muscle atrophy in tubercular joint disease is *progressive* if the disease be not checked, and *permanent* if the disease be permitted to go beyond a certain point. This can be abundantly proved from ordinary clinical experience. Every one knows how rapidly progressive muscle atrophy is in tubercular and in some other joint diseases. Unfortunately the atrophy does not limit itself to the muscles, but extends to the bones and to the limbs as a whole.<sup>5</sup> Now, this is just the result one might expect from diminished blood-supply and consequent deficient nutrition. That such atrophy is apt to persist is also easily proved from ordinary clinical experience. Do we not see everywhere shrunken and distorted limbs, the result of untreated and neglected tubercular joint disease? Such atrophy is apt not only to persist, however, but to progress, as in the case of the limb which I show you this evening, in which the femur and tibia have become (from tubercular disease of the knee joint) about half the size (in bulk) of the corresponding bones of the other

<sup>1</sup> *Lancet*, May 30 and June 6, 1893.

<sup>2</sup> *Diseases of Nervous System*, New Sydenham Society, 1889.

<sup>3</sup> *Loc. cit.*, p. 23.

<sup>4</sup> *Loc. cit.*, p. 27.

<sup>5</sup> Charcot says that atrophy started by joint disease is apt to persist, and to cause general atrophy of the limb, *loc. cit.* (p. 26).

limb, whilst their length remains much the same; and this change has taken place in twelve months, showing that the comparative alteration in size cannot be the result of mere want of growth on the diseased side.

Now, it seems to me that we have hitherto had no adequate explanation of such a state of matters as this. Why should such a simple atrophy steadily, and sometimes rapidly, increase? Why should it persist, and sometimes go on to permanent and progressive wasting of a whole limb?<sup>1</sup>

That such an arterial contraction exists in stumps after amputation has been proved by Thoma.<sup>2</sup> He has in such cases found a permanent and progressive arterio-stenosis, which is due to two causes—1, Concentric atrophy of the media; and, 2, thickening of the intima by deposit of connective tissue. If this or something similar could be found in connexion with tubercular joint disease, it would abundantly explain and clear up the cause of the progressive and permanent muscle atrophy.

Dr Bruce has kindly examined for me specimens of the popliteal artery and a portion of muscle from the limb which I have shown to-night, and he reports that he has found slight sclerosis of the intima in the artery and no degeneration in the muscle. There has not been time, however, to make an exhaustive examination, but Dr Bruce promises to carry out the investigation more fully if I can supply him with material, which I hope to be able to do.

5. I would now go a step further, and say that it is probable that tubercular joint disease causes muscle atrophy by producing anæmia of the muscle through contraction of the arteries.

In support of this statement I would mention one or two facts which I think bear on the subject—

*a.* Tubercle is associated with anæmic conditions. The so-called cold abscesses are known to be tubercular, and they have very little vascularity or inflammatory action about them—hence their name. Again, tubercular joint affections are so bloodless that they used to be called “white swelling.” As one more surgical illustration, I would point to the tubercular necrotic areas which we find in bones, which are absolutely bloodless and apparently brought about by destruction of the blood-supply. I do not say that all these conditions are due to arterial contraction, for I do not know; but I point to the association of these anæmic conditions with tubercular disease. I have often thought that in this local anæmic condition of tubercular parts there is an illustration of Nature trying to produce a cure by shutting off the blood-supply: in some cases succeeding, in others failing, through falling into a Scylla in avoiding a Charybdis. In other words, the anæmia,

<sup>1</sup> I have seen cases where the general limb atrophy was so great, and evidently progressive, as to necessitate amputation even after the joint disease was cured.

<sup>2</sup> *Loc. cit.*, p. 301.

whilst checking the inflammation, possibly fails to cure the disease by not supplying sufficient blood to destroy the bacilli.

*b.* My next point is that irritation, especially a slight but constantly acting irritation, causes contraction of bloodvessels. Now, tubercular irritation is of this kind, and Charcot says that diseased joints cause rapid and early muscle atrophy, because the irritation is continuous.

*c.* Another point is that congestion is unfavourable to tubercle. Rokitansky stated long ago that "a congested lung possesses an immunity against tuberculosis," and it is admitted that there is some truth in this statement. Page suggested that Koch's tuberculin acted in the same way; and Bier founded his method of treating tubercular affections by congestion on this dogma of Rokitansky's.

*d.* My next point I submit with considerable deference, because it is a mere fancy of my own. It is that possibly the products of the tubercle bacilli tend to cause vaso-contraction, just as the products of pyogenic organisms cause vaso-dilatation and diapedesis. Ogston, Watson Cheyne, and Treves hold that tubercular abscesses contain no true pus and few leucocytes. My friend Mr Stiles tells me, however, that more recent investigations show that there is very little difference between the contents of a tubercular abscess and any other collection of pus. That may be so, so far as the microscope is concerned, but my clinical experience tells me that there is a considerable difference. And I still think that some variation in the vascularity may account for this difference.

6. I now come to my last point. When I first thought of a definite relation between the muscle atrophy and the arterial contraction which I saw in cases of tubercular disease of joints in the way of cause and effect, I realized that there must be some definite connexion between the vessels and the muscles, either directly or through the nervous system. It occurred to me also that the tendency of tubercular joint disease to pick out certain muscles for the manifestation of atrophy would perhaps help me. I found, however, that whilst in some joints (the shoulder, for instance) association of the diseased joint and the atrophied muscles could be made out quite easily, both through the nervous and the vascular systems, in other joints (the knee, for example) the relation was round about and difficult to trace. Arguing, however, from the joint disease as the primary cause, I could see how arterial contraction could be produced quite easily, provided the influence passed through the vaso-motor system, for the vaso-motor system is freely and copiously connected with the spinal system of nerves.

Next came the connexion between the vessels and the muscles, and that, of course, was easily made out. But, as I have already indicated, in this connexion a very significant fact cropped up as likely either to support or destroy my line of proof—viz., the

constant and prominent selection of certain muscles for the exhibition of the atrophy in connexion with tubercular joint disease.

Ferrier<sup>1</sup> tells us that extensor muscles atrophy much more quickly than flexors, because they are weaker. One might suppose, therefore, that given a general atrophy from contraction of the main artery of the limb, the extensors will manifest the change first and most. But there is something more. There is always a close relation between the special vessels supplying a given joint and those supplying the muscles that atrophy most. (See Appendix.) From this intimate relation I gather, therefore, that the special atrophy of certain muscles can be explained on the diminished blood-supply theory.

Now, I am quite aware that there are other possible ways of turning the argument according as one looks at it. For instance, it might be said that whilst the joint disease is, as before, the primary factor, the vaso-stenosis is not the cause of the muscle atrophy, but its result. This would be in keeping with Thoma's theory<sup>2</sup> that the demand creates the supply, and I thought at one time that this might be the true explanation—viz., muscle atrophy first, then arterial contraction and general progressive atrophy of the limb, but I had two difficulties:—1. This theory left the muscle atrophy unexplained, except by a vague and mysterious trophic nervous influence. 2. The muscle atrophy has been shown to be simple, and not such as occurs in connexion with nerve causes. In other words, the facts known fit in better with the explanation that the arterial contraction causes the muscle atrophy than with any other theory I know of.

I would sum up my argument as follows:—

1. Muscle atrophy is a constant and prominent symptom in tubercular joint disease.

2. No explanation of this atrophy has been suggested hitherto, except a vague and mysterious reflex influence.

3. Contracted arteries have been seen and proved to exist.

4. The muscle atrophy is of a kind likely to be caused by deficient blood-supply rather than by nerve influence.

I assume, therefore, that arterial contraction and consequent diminished blood-supply is the cause of muscle atrophy in tubercular joint disease.

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#### APPENDIX.

*Relation of Arteries supplying Joints to Muscles which Atrophy.*

*Shoulder.*—Posterior circumflex and supra-scapular arteries supply joint and also deltoid and scapular muscles.

*Elbow.*—General arterial anastomosis round elbow, formed by branches of superior profunda, inferior profunda, and anastomotica.

<sup>1</sup> *Lancet*, 12th August 1893, p. 386.

<sup>2</sup> *Loc. cit.*, pp. 265-269.

Triceps, brachialis anticus, and coraco-brachialis supplied by superior profunda; biceps by special branches from brachial artery; it sometimes holds out well.

*Hip.*—Branches from gluteal and sciatic arteries supply the joint, and also muscles of hip. Artery to round ligament comes from internal circumflex of profunda; hence the thigh muscles atrophy also.

*Knee.*—Free anastomosis between articular branches of popliteal, branches of profunda and anastomotica magna. Quadriceps supplied by various branches from superficial femoral, profunda, and anastomotica, and also branches from superior articular arteries.

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*The Vice-President* said they would be glad to hear remarks on Mr Miller's paper.

*Mr Cathcart* said that as Mr Miller had mentioned his name in speaking of one of his lines of argument he might perhaps say a word or two about this very interesting paper. He personally did not feel that they had worked out the possibilities of non-use, and until they had fully worked that out he did not think they need bring forward a reflex vaso-constriction. As to non-use, what he meant was this,—That when any part of the body was affected which had a sensory supply, there seemed to be a reflex inhibition of that part through the nervous system. Physicians would probably corroborate this when he asked if it was not the case that when a patient had pleurisy or pneumonia, that side of the thorax did not move with the same freedom as the opposite side; or that in peritonitis the abdominal respiration was affected. The movements of a joint, too, were affected when the joint was diseased. This reflex condition was not one that the patient was necessarily aware of, nor was it under control. A child lame with tubercular knee joint might at first suffer nothing, and was often not aware of anything wrong; but the surgeon found wasting of muscle and a stiff joint. He thought that until they had excluded this possible effect of reflex inhibition of the muscles acting on this joint and associated with it, they did not require another theory. At the same time he thought it required a great deal of very minute observation to be quite sure about reflex want of use. If a healthy joint was put on a splint, there being no reflex inhibition the patient moved it when he could. Anything that brought on reflex vital inhibition was more efficient for disuse than an actual splint. With regard to the question of arterial contraction, it seemed to him excessively difficult. If a patient had an ankylosed or amputated limb, apart from any disease the muscles at once atrophied, and so did the arteries, *e.g.*, after Syme's amputation there was no further use for the gastrocnemius, and all the muscles and arteries from the knee downwards atrophied without any possibility of reflex affection by any inflamed part. One other



point. Mr Miller said that irritation caused arterial contraction. Two or three difficulties were involved in that. The irritation was in the joint, but the contraction was in connexion with muscles. There was nothing to show that the irritation had any effect on the muscles at a distance from the joint, when it did not produce contraction. One certain thing in connexion with inflammation was that the vitality of the tissues was interfered with,—*i.e.*, the tissues were irritated, and one of the first changes was dilatation of bloodvessels, and Mr Miller himself said that septic inflammation produced congestion, but tubercular irritation produced contraction. Now, septic poison was as much an irritant as tubercular. One other difficulty was that he (Mr Cathcart) was not quite sure that tubercular joints were so very anæmic. One of the symptoms they trusted to was called "white swelling." Relative to other forms of swelling it might be so. But another of the symptoms they trusted to was heat of the joint as compared with that of the opposite side. If one examined a recently amputated joint one was struck with many parts of it being unduly vascular. But he knew it was very easy to criticise, because the subject was exceedingly difficult. He thought they were much indebted to Mr Miller for having brought such a thoughtful and interesting paper before them.

*Dr Gibson* said there was just one question he was going to address to Mr Miller, and that arose out of a point which had just occurred to him with reference to this very interesting paper. He had personally observed that while in cases like that of this youth's leg there was atrophy of osseous textures as well as of soft parts, if they happened to have a tubercular affection of the sheaths of the tendons, for instance, they had a very considerable amount of atrophy of muscles, but no atrophy of bones at all. He would like Mr Miller to explain that conundrum to them, because it seemed to him it bore on this point. He was sure any one could vouch for the accuracy of that statement. He would be inclined to explain it in this way, along the lines laid down by a paper of the late lamented *Dr Ross* of Manchester on the segmental distribution of the nervous system. He thought it was extremely probable that the effect was produced by irritation of some sort, probably arising from a toxin formed by tubercle bacilli. That this was in accordance with the theory of the segmental distribution applied to every texture implicated,—in the one case bones and muscles, in the other muscles alone affected. That, it seemed to him, might possibly throw a little light on this condition. Without trespassing further on the time of the Society, he wished to bring it to Mr Miller's attention.

*Mr Miller*, in reply, said that with regard to Mr Cathcart's remarks he had tried to guard himself. He had said that he did not look upon this vascular contraction as the only cause. It did not exclude want of use. In fact he mentioned as one of *Thoms*

factors the want of use. But he had noticed a fact which showed that want of use could not be the cause of muscle atrophy in tubercular joint disease. He had frequently seen cases of tubercular joint disease with atrophy that had distinctly improved inside a plaster case. The muscle atrophy had certainly not got worse. In fact, in some cases he thought the muscles showed improved nutrition,—*i.e.*, provided the joint got better. As regards the reflex theory, he took it for granted that it was admitted that atrophy was due to reflex action. Charcot took it for granted all through his lectures. And, curiously, he got quite a new light on reading Charcot's remarks on the subject. Charcot attributed the discovery to John Hunter. He had been looking up John Hunter's works, but could not get any reference to it. With regard to the other point, of course dilatation of the bloodvessels took place afterwards; but Thoma, in referring to this subject, spoke about contraction occurring as the result of irritation, and Charcot spoke of joint disease causing atrophy. He (Mr Miller) was piecing the two things together—perhaps some might think unfairly. He did not say that tubercular disease caused anæmia, but was associated with anæmia. Of course there was vascular dilatation in every inflammation. He thought Mr Cathcart's remarks might explain Dr Gibson's. The reason why atrophy occurred in tubercular affections of sheaths of tendons was that the muscles were so very thoroughly thrown out of action. They had to be completely rested. At the same time there might be some toxic effect of the tubercle bacillus products.

## 2. CASE OF DEEP-SEATED TROPICAL ABSCESS OF THE LIVER, TREATED BY TRANSTHORACIC HEPATOTOMY. RECOVERY.

By BYROM BRAMWELL, M.D., F.R.C.P. Edin., Lecturer on Practice of Physic, Assistant Physician, Royal Infirmary; and HAROLD J. STILES, M.B., F.R.C.S. Edin.

### A.—THE MEDICAL HISTORY OF THE CASE AND DIAGNOSIS BY Dr BYROM BRAMWELL.

G. G., a strong muscular man, aged 28, was seen, with Dr Curtis Whyte of Dalkeith and Dr T. W. Dewar, on 8th October 1895.

*Previous History.*—The patient stated that he had enjoyed perfect health until about six years ago, when he suffered from a rather severe attack of gonorrhœal arthritis, affecting especially the knee, both ankles, and the toes. During this illness he was confined to bed for nearly a month, and was afterwards treated for some weeks at Buxton. The following spring he again suffered from rheumatism in both ankles, and was again treated at Buxton. The rheumatism then gradually left him, and he has been free from it ever since.

In June 1894 he went to Lagos, on the West Coast of Africa. He describes Lagos as a flat, swampy, stinking, fever-breeding place, with no safer water-supply than the rain collected from the roofs of the houses. A month after reaching the colony he was seized with fever of a malarial type; the febrile attacks recurred at longer or shorter intervals, and usually lasted for two or three days at a time.

In April of the following year (1895) he was seized with a severe attack of dysentery. This necessitated his removal to hospital, where he remained three weeks. From hospital he was conveyed direct to the ship which brought him home. He derived great benefit from the voyage, and states that on his arrival in this country in June 1895 he was perfectly well.

He remained in good health until the end of August, when, while away from home on a holiday, he was attacked with severe dysenteric diarrhœa. He returned home looking and feeling ill.

He was seen by Dr Whyte on September 4. The diarrhœa soon yielded to treatment, but some days afterwards he began to complain of intermittent pains in the right shoulder, and of a catching pain in the right hypochondriac region—at a point corresponding to the junction of the sixth and seventh right costal cartilages with the sternum. The pain in the hypochondriac region was increased by taking a long breath, laughing, or yawning. It was so much aggravated by laughing that he was obliged to get up and leave the room whenever there was any joking going on. This pain continued notwithstanding treatment.

On the evening of September 20 he was seized with a chill while out walking, and on his return to the house he had a distinct rigor. The temperature ran up to  $103^{\circ}$ . The rigor was followed by profuse perspiration. He was now kept in bed and placed on quinine—five grains four times daily. But as the febrile disturbance persisted in spite of the quinine, and as the patient complained on several occasions during the next fortnight of slight chilliness not amounting to distinct rigors, Drs Whyte and Dewar began to suspect that he was probably suffering from an abscess of the liver, although no local signs of such a condition were discoverable. During this period night sweats were frequent; the temperature always ran up to  $101^{\circ}$  or  $101^{\circ}5$  at night, but was usually normal in the morning; the appetite became poor, but the tongue remained clean; there was some loss of flesh.

*Present Condition.*—On October 8 I was asked to see the patient in consultation with Drs Whyte and Dewar. The most careful examination failed to detect any physical evidence of local disease. The abdominal and thoracic organs all appeared to be normal. The liver was not enlarged, and there was no localised tenderness even on firm pressure over any part of the organ.

The pulse frequency was only slightly increased; the respirations numbered 16 per minute; the temperature at the time

of my visit was slightly below the normal; the tongue was clean; the appetite poor; the bowels regular. The patient had, since his illness commenced, got a little thinner, but there was certainly no emaciation. The evening temperature had for some weeks previously been distinctly elevated; there had been several attacks of chilliness, followed by sweating, and one definite rigor.

Notwithstanding the absence of any enlargement of the liver and of any tenderness on pressure over the organ, I agreed with Drs Whyte and Dewar in thinking that the patient was in all probability suffering from an abscess of the liver. This opinion was based on the following line of argument:—(1) That the patient was suffering from fever of a hectic type, gradual and progressive emaciation, and occasional attacks of chilliness, hardly amounting to distinct rigors; (2) that the febrile disturbance was irregular in type (in other words, not distinctly malarial), that it was uninfluenced by quinine, and that there was no definite enlargement of the spleen; (3) that the patient had suffered from severe tropical dysentery; (4) that the patient complained of pain in the right shoulder and of a catching pain in the right hypochondriac region at the junction of the sixth and seventh costal cartilages with the sternum; (5) that the most careful examination failed to detect any evidence of disease in the lungs, heart, or any other organ, capable of accounting for the symptoms; and (6) that there was nothing, either in the personal condition of the patient, his previous state of health, or the family history, suggestive of tubercle—acute tuberculosis being one of the conditions which was naturally thought of as a possible cause of the symptoms.

For the reasons given above, I concluded—

*Firstly*, that the febrile disturbance was not due to malaria, to tubercle, nor to ulcerative endocarditis; further, there was no suspicion of typhoid fever.

*Secondly*, that it was apparently due to some internal suppuration.

*Thirdly*, since there was no discoverable local lesion in any other organ (*negative evidence*), and because (*a*) of the very definite history of dysentery, and (*b*) of the presence of pain in the right shoulder and right hypochondrium (*positive evidence*), that (notwithstanding the absence of any obvious enlargement of the liver) the internal suppuration, which we believed to be present and to be the cause of the fever, was in all probability situated in the liver.

Believing, then, that the case was one of hepatic abscess, we decided that, if the symptoms did not subside after further treatment with still larger doses of quinine, the liver should be explored by diagnostic puncture, with the object of endeavouring to localise the pus.

October 23.—As no improvement had resulted, Mr Stiles was asked to see the case. He agreed with the diagnosis, but considered it advisable to wait for further indications of the position of the supposed abscess before proceeding to operative interference.

On October 30 another consultation was held (Drs Whyte, Dewar, Bramwell, and Mr Stiles being present). The patient was evidently losing ground; his expression was indicative of languor and debility; his features were somewhat pinched and shrunken; he was thinner; the fever was higher; and although there was no jaundice, he exhibited in some degree the sallow, earthy complexion which is frequently observed in patients suffering from hepatic abscess.

He complained of a dull, ill-defined pain and sense of weight over the right hypochondrium; of attacks of chilliness, amounting almost to rigors; and of profuse perspirations, especially during sleep. He preferred a dorsal decubitus, but it could not be said that lying upon his left side gave rise to any additional discomfort.

The temperature chart during the previous fortnight showed a distinctly hectic type of fever, the evening rise ranging from  $102^{\circ}$  to  $103^{\circ}$ , with a morning fall to between  $99^{\circ}$  and  $100^{\circ}$ . The tongue was moist, with a white fur on the dorsum, and red edges. The appetite was markedly impaired, but there had never been any vomiting. The bowels were normal. The urine was high coloured and loaded with urates, but otherwise normal.

The abdomen was flat and flaccid. The epigastrium was, if anything, a little hollowed out; its two halves were perfectly symmetrical, and moved equally with respiration. The muscles attached to the right costal margin were free from any rigidity. Two of the gentlemen who took part in the consultation thought that there was just a suspicion of greater fulness on the right side opposite the seventh, eighth, and ninth ribs in the mid-axillary line, but the alteration, if any, was so very slight (and two of us were not satisfied that it actually did exist) that little or no significance could be attached to it. The intercostal spaces over the region of the liver were quite distinct, and there was no trace of subcutaneous œdema. The patient was unable to refer to any distinct local pain further than a sense of weight and aching over the whole of the right hypochondrium, and of a catching pain on taking a deep breath, at the junction of the sixth and seventh right costal cartilages with the sternum. There was no local tenderness; indeed, the ribs and interspaces could be very firmly pressed upon without causing any discomfort. There was certainly no enlargement of the liver in a downward direction, and percussion failed to show any evidence of a dome-shaped enlargement upwards. There was no cough. The lungs appeared to be quite healthy. At the extreme base of the right lung, in the posterior axillary line, a very faint friction rub (probably sub-diaphragmatic) was heard over a very localised area about the size of a five-shilling piece. There was no enlargement of the spleen. The heart was quite normal. Rectal examination was entirely negative.

The general condition was less satisfactory than it was when I

had last seen the patient a fortnight previously. I saw no reason to modify my previous conclusion that the cause of the febrile disturbance was in all probability suppurative hepatitis, and that the pus was probably deeply seated in the upper and back part of the right lobe. The very localised friction rub over the base of the right lung, which was the only physical sign indicative of disease, seemed to support this opinion, which was concurred in by all the gentlemen who were present at the consultation, and was communicated to the patient's relatives.

The question of immediate exploratory puncture was again debated, and it was decided to wait for a few days longer, with the object of seeing if any further symptoms or signs indicative of the position of the supposed abscess would develop; and then, whether such development took place or not, that the operation of exploratory puncture should be performed.

*B.—THE SURGICAL TREATMENT OF THE CASE, AND REMARKS BY MR STILES.*

On November 7 I was sent for to see the patient again. He had had a bad night in consequence of having been seized with a lancinating pain, localised to the seventh interspace in the mid-axillary line. The evening temperature during the previous week had risen to between  $103^{\circ}$  and  $104^{\circ}$ , and on one occasion reached  $104^{\circ}6$ ; there had been no distinct rigors, but the night sweats were more profuse. As the temperature had gradually been getting higher and the patient slowly but surely losing ground, it was decided to explore the liver for pus, notwithstanding the absence of any enlargement. A medium-sized trocar and canula belonging to the ordinary Potain's aspirator was pushed into the liver in the mid-axillary line through the seventh intercostal space; this situation was selected, firstly, because from it the upper and back part of the right lobe could be most readily reached, and, secondly, because it was the seat of the sharp localised pain of which the patient complained. Pus was reached after the trocar had penetrated for a distance of about  $2\frac{1}{2}$  inches from the surface, a slight resistance being encountered as the wall of the abscess was pierced. About a tablespoonful of brick-red pus, strongly suggestive of anchovy sauce, was removed, and received into a sterilised bottle. It was found that the canula could be pushed up to the hilt without meeting with any resistance, thus proving that the abscess was of considerable size. The pus, which was without odour, was carefully examined microscopically for organisms, but none could be discovered. Gelatine and agar-agar tubes were inoculated, and remained sterile. In view of the fact that amœbæ have been found in such abscesses by Kartulis, Koch, Osler, Councillman and Laffleur, Galloway and others, many films

were specially stained for them, but with a negative result. The "pus" consisted chiefly of granular detritus, fragments of chromatin, pigment granules, and a few red and white blood-corpuscles.

On November 9, assisted by Mr Ernest Fortune, I performed the following operation:—The skin having been pulled downwards, an incision 4 inches in length, and commencing in the mid-axillary line, was carried obliquely downwards and forwards over the eighth rib, and a portion of the latter,  $2\frac{1}{4}$  inches in length, was excised subperiosteally. The upper edge of the wound was then retracted and a corresponding portion of the seventh rib was excised, also subperiosteally. With an anemism needle a catgut ligature was tied round the extremities of each of the periosteal troughs which remained, thus securing the intercostal vessels. The periosteum which intervened between the ligatures was then removed, as also were the adjacent intercostal muscles and a digitation of the serratus magnus. In this way an area of costal pleura, about 2 inches square, was exposed, an endeavour having been made not to wound the pleura in dissecting the above-mentioned structures from off it. It was found, however, that a small opening had been made into the pleura in dividing one of the ribs, the result being that a small quantity of air could be heard to enter the chest with each inspiration. The general cavity of the pleura was next shut off by the introduction of a circle of interrupted and overlapping catgut sutures, according to the method recommended by Mr Godlee,<sup>1</sup> but deeply enough to penetrate not only the costal and diaphragmatic pleura, but the whole thickness of the diaphragm along with the capsule of the liver. A crucial incision was then made through the costal pleura within the area enclosed by the sutures, and the diaphragm, covered by its pleura, exposed. The diaphragm was then incised parallel to its fibres, and—as was anticipated from the slightly thickened and opaque condition of its pleura—was found to be adherent to the liver. As, however, the adhesions were recent, and allowed the fibres of the diaphragm to be readily separated from the capsule of the liver, these two structures along with the four doors of the costal pleura were united by a second continuous circular suture placed within the interrupted ones already mentioned. An area of liver about the size of a half-crown piece was thus exposed within the second or inner circular suture. The circumference of the wound was then packed with a strip of gauze, and the largest trocar and canula of the aspirator was pushed backwards and slightly upwards into the liver, and the abscess reached at a distance of about 1 inch from its surface. With the canula retained in position to act as a guide, a knife was pushed alongside it into the abscess, and the opening dilated by a pair of dressing

<sup>1</sup> *Brit. Med. Journ.*, Oct. 22, 1887.

forceps so as freely to admit the finger. There was no hæmorrhage to speak of. Before allowing the contents to escape, the finger was used to ascertain the size and nature of the abscess cavity. It was found that we had to deal with one large cavity, nearly the size of a child's head, situated in the upper and back part of the right lobe—the commonest situation. The finger was unable to reach the limits of the abscess until the greater part of the contents had been allowed to escape. The wall of the abscess was soft and friable. There were no diverticula. The pus, which had the appearance already described, was of a mucoid consistency, and contained much debris and soft pulpy necrotic masses. After evacuating the contents, the cavity was flushed out with a large quantity of sterilised water, which brought away a further quantity of necrotic tissue. The cavity was drained by means of a Keith's glass tube, 3 inches long, and fitting tightly within a flanged rubber tube long enough to project for an inch or so beyond the glass. In this way collapse of the tube was prevented, while at the same time the risk of its extremity pressing injuriously upon the wall of the abscess was avoided. The wound was stuffed with iodoform gauze, and partly closed by a couple of sutures introduced at each extremity.

*Progress.*—*Nov. 10.*—Slept well; temperature normal; pulse 66; respirations 26; sweating much less. Complains of pain when he tries to move arm.

*Nov. 11.*—Patient looks much brighter, having already lost to a great extent the earthy and pinched look. Pulse and temperature normal; more inclined for food. Dressing soaked with a considerable quantity of dark red muco-pus, almost gelatinous in consistency.

*Nov. 13.*—Been perspiring more freely; temperature  $101^{\circ}$ ; pulse 80; respirations 28. Dressed; tube found to be blocked with gelatinous-looking necrotic masses. Retention of 2 or 3 ozs. of muco-pus, which accounts, no doubt, for the elevation of temperature. Iodoform gauze-stuffing removed and renewed. A probe passes for 5 inches before striking the further side of the abscess.

*Nov. 15.*—Temperature been normal since last dressing. Discharge copious and of same character. Appetite much improved; can move arm more freely.

*Nov. 17.*—Discharge same character, but less in quantity.

*Nov. 21.*—Complained of pain in right side since last night. This found to be due to the pressure of the flanged edge of the glass tube, which now passes very obliquely upwards and backwards. Œsophageal tubing substituted for the glass one.

*Nov. 24.*—No pain since tube changed. The discharge is now very scanty, and has quite altered its character, being ordinary or laudable pus, such as one would expect to come from an abscess cavity lined by healthy granulation tissue. The unhealthy necrotic lining has evidently been completely cast off. The probe passes



very obliquely upwards for about 3 inches ; from its direction it is evident that contraction is taking place mainly from below, in consequence, no doubt, of the upward pressure exerted by the abdominal contents.

*Nov. 24.*—Smaller and shorter tube introduced.

*Nov. 29.*—Tube removed ; the sinus only  $1\frac{1}{2}$  inch in length ; stuffed with iodoform worsted.

The further progress need not be detailed ; suffice it to say that the patient rapidly gained in weight and strength, so that by the end of the fourth week he was able to be out of bed, and three weeks later he was going about out of doors with the wound completely healed.

On January 2, 1896, physical examination showed that the liver was very considerably diminished in volume ; the relative dulness in the mammary line commenced at the upper border of the fifth rib and became absolute at the sixth interspace ; the lower edge did not quite reach the costal margin, and light percussion gave a stomach note over the entire costal angle right up to the sternum. On the right side, below the angle of the scapula, and in the lower axilla, the lung note was somewhat impaired, and ceased altogether two finger's breadth above that on the left side. The breath sounds over this area were feeble, otherwise normal. There was no friction. There was some falling-in of the chest wall on the right side opposite the seventh, eighth, and ninth ribs, but the expansion of the chest was equally good on the two sides.

The patient was seen again a few weeks ago, and appeared to be in perfect health ; he has gained two stones in weight since the operation, and is in the habit of taking a great deal of exercise ; his digestion leaves nothing to be complained of. The cicatrix is somewhat depressed, and becomes drawn in during inspiration. The physical signs are much the same as they were five months ago. The whole of the costal angle is still free from liver dulness, but the breath sounds at the base of the right lung are more distinct, and can be heard almost down to the same level as on the left side.

*Remarks.*—In reviewing this case from the surgical aspect, there are one or two points to which further allusion may be made.

Although the history, the discomfort in the hepatic region, the constitutional symptoms, and the healthy condition of the other organs, all pointed to suppurative hepatitis, it nevertheless remained for the aspirator to establish the diagnosis, to determine the situation and size of the abscess, and to decide as to the nature of the operation which would require to be performed.

If pus be present in the liver without giving rise to any external manifestations, then we must conclude that we have to deal either with small multiple abscesses or with a single abscess,

which, if of considerable size, will in all probability be situated either in the upper or upper and back part of the right lobe. The question therefore arises as to the best site from which to make an exploratory puncture in order to reach the pus by the safest and most direct route. As the result of investigations in the post-mortem room, I came to the conclusion that the seventh intercostal space in the mid or anterior axillary line should be the point first chosen, and the trocar passed either vertically to the surface, or with more or less inclination upwards and backwards. The lower part of the pleural cavity, if not obliterated by adhesions, will of course be traversed, but the lower edge of the lung will be above the puncture. Should this fail to strike the pus, further exploration should be made through the seventh space in the scapular line, and, if necessary, from the sixth and eighth spaces. If there be no downward enlargement of the liver, the ninth space would be too low, and there would be considerable danger of the trocar entering the colon, the gall-bladder, or even the duodenum.

The next question to consider is, Having struck the pus, should the operation be at once proceeded with? I think not. A small quantity of pus should be withdrawn into a sterilised bottle and subjected to careful microscopic examination, and culture tubes should be inoculated. By this means, the surgeon, within forty-eight hours, will gain information as to whether the abscess be sterile, or whether organisms be present, and if so, their nature. In a large proportion of cases, the pus in tropical abscesses of the liver has been found to be sterile. In other cases it has contained the *amœba coli*, either alone or in conjunction with the ordinary pus-forming organisms, or the latter only have been found. Should pyogenic organisms be discovered, then the subsequent operation must be performed in such a way as to prevent infection of the pleural and peritoneal cavities. This, of course, is best done by operating in two stages. The first operation consists in the exposure of the liver, the shutting off of the serous cavities by sutures, and the plugging of the wound to bring about adhesions; at the second, a few days later, the abscess is opened. If, however, the pus be sterile, as it very frequently is, then I see no reason why the abscess should not be opened at once, and the patient thereby relieved from the anxiety and shock of a second operation.

Even although the pus be sterile, that should not prevent the operator from shutting off the pleural and peritoneal cavities as far as possible. To do this, portions of two ribs should be excised, and a good large area of costal pleura exposed. Having excised the ribs, the soft parts (periosteum, intercostal muscles, and a digitation of the serratus magnus) should be dissected off the costal pleura, if possible, without wounding it, a step which will require considerable care when dealing with thin healthy pleura. From the reports of cases to which I have had access I find that

the costal pleura has been incised before stitching it to the diaphragm. It appears to me that it is a much better plan to pass the sutures before incising the pleura; with the costal pleura freely exposed, and stretched across the floor of the wound, the suturing of its circumference to the diaphragm and liver is rendered simpler and more satisfactory than would be the case if the pleural cavity were first opened into. When the right lobe of the liver is the seat of an abscess, the diaphragm is pushed up into close contact with the costal pleura, so that with a fully curved needle (a Hagedorn's and holder) a circle of interrupted and overlapping sutures may readily be introduced. Most surgeons, I observe, have introduced them merely into the diaphragm; but seeing that we can never be certain whether the liver has become adherent to the diaphragm, it is better to carry the sutures through the diaphragm into the capsule of the liver, and in this way to shut off the pleural and peritoneal cavities at one and the same time. Another advantage of not incising the pleura until after the sutures have been introduced, is that very little air enters the chest, and complete and sudden collapse of the lung is thus prevented—a matter of some importance with the patient under the influence of an anæsthetic. In our own case, air was sucked into the chest through a small accidental opening into the pleura as well as each time the pleura was punctured in the act of introducing the sutures. Dr Whyte, who administered the anæsthetic, was unable to observe that this produced any effect upon the respiration. The same observation has been made when the pleura has been wounded in the act of removing a cervical rib.

The inner continuous circular suture may, perhaps, not have been necessary; as, however, the adhesions between the diaphragm and liver were of recent origin, it was thought safer to employ it; in the absence of such adhesions it should certainly be used.

Special precautions have been taken in order to diminish the amount of hæmorrhage resulting from the necessary incision into the liver to reach the abscess. Zancarol,<sup>1</sup> who has probably had the largest experience in the treatment of hepatic abscess, recommends that the cautery be used for this purpose. Edmunds,<sup>2</sup> having introduced a special trocar and canula into the abscess, withdraws the former, and substitutes for it a narrow grooved director; the canula is then withdrawn, and a long wedge-shaped knife passed along the director into the abscess.

Except in abscesses which are unusually deep-seated, the simple method of pushing the knife into it alongside an ordinary canula left in position to act as guide, and following up this with dressing

<sup>1</sup> *Traitement chirurgical des abcès du foie des pays chauds.* Paris, Steinheil, 1893.

<sup>2</sup> *St Thomas's Hospital Reports*, new series, vol. xxi., 1893, p. 205.

forceps to dilate the opening sufficiently to introduce the finger, appears to be a perfectly safe procedure.

As regards the treatment of the abscess cavity, Fontan<sup>1</sup> recommends that its wall be scraped, and claims that by this means the healing process is hastened. In a large abscess of the liver such as has been described, thorough douching, followed by free drainage, is simpler, and gives perfectly satisfactory results, and, moreover, is free from the risk of hæmorrhage or of opening into the biliary passages.

The last point to which it is necessary to refer is one which has already been mentioned,—namely, that cicatrization of the abscess takes place chiefly from below. This is a point which should be borne in mind in considering the level at which the abscess should be opened, as it is evident that drainage will be better maintained if the abscess be opened at its upper rather than at its lower part.

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*Surgeon-Major Black* said that some time ago he met with a case of an invalid soldier very similar. It was in the days before antiseptics and drainage-tubes were in use. It was a large abscess, very chronic, diagnosed beforehand in consultation. There was protrusion of right hypochondrium and between the ribs as well, the liver coming below the cartilaginous border. There was scarcely any doubt about the nature of the disease, and, after due consultation, operation for evacuation was decided upon, and he introduced a canula below the border of the cartilages into the cavity in the liver, and in due course followed a flow of pus through the canula. This part of it was altogether very successful, and everything went right for a few days. The canula was inspected every day. The flow of pus went on regularly. At last events took a turn, and had there been antiseptics in those days, a drainage-tube and dressings would have been of service. The hard canula was still kept in, tied down by tape, and so on. By-and-by pus began to ooze along outside of the canula. They thought peritonitis was impending. This turned out to be the case, and the patient died of peritonitis. There was no superficial hepatitis, no costal pleuritis. They had not reckoned on non-adhesion of the liver all over it. Pus therefore had escaped into the peritoneum alongside the tube. In this case the abscess was more towards the border of the liver, so that it had easy access to the peritoneum. Military surgeons in India had these cases very frequently, and nowadays it was a very successful operation under antiseptics.

*Dr Leith* said he would like to convey his congratulations to Drs Bramwell and Stiles for bringing forward such an excellent illustration of the more fortunate results which one now occasionally met with, more frequently than in the past, in cases of hepatic

<sup>1</sup> *Rev. de Chir.*, Feb. 1892.

abscess. Few of them could presume to have had much experience in this country of this rare condition. At the same time, the experience of the past and the literature were always open to them, and therefore they were at liberty to judge of this case according to these lights. He thought they would agree that nothing had been left undone in the diagnosis and treatment of this case. He might ask Dr Bramwell if, in excluding malaria, he had examined the blood for organisms, as that was necessary besides the negative point, viz., the absence of benefit from quinine. He should like to allude to the increase in medical knowledge within recent years with regard to hepatic abscess. The books of a few years ago told them that hepatic abscess, when treated artificially, was not nearly so successful as when allowed to find a drain for itself; and further, that when pus was formed the time for active interference was gone by. When they found such extraordinary statements made by Indian surgeons, to whom they looked for information on the subject, they were delighted to find justification for the newer progressive statements now made, to the effect that hepatic abscess taken as early as in this case was not such a serious condition. He (Dr Leith) believed that quite 50 per cent. were followed by recovery. This case was a perfectly classical one in almost every point. The early resort to aspiration was to be highly commended. He had spoken to an Indian surgeon—he was sorry he did not see him there to-night—on this question, who told him he never failed to aspirate early. When he excluded other lesions, he never failed to use something like 6 feet of aspirating needle before giving up the search for pus. He selected the seventh or eighth interspace, usually in the anterior axillary line, entered the aspirator inwards, then turned it upwards. If he found nothing, he re-inserted it in various directions before complete withdrawal, so that there was only one puncture through the chest wall and capsule of the liver. If he still got nothing, he tried the left lobe from the front in the same way. Dr Leith illustrated the procedure by sketches on the black board. That was a pretty extensive procedure, and he said he never had any harm resulting from it. There was no doubt that the only danger was hæmorrhage. That had happened, and was not to be entirely excluded. Only the superficial and not the interior hæmorrhage was to be feared. So far as the destruction of liver substance was concerned, they had far more liver than they needed, and could quite afford to destroy as much as that without any harm whatever. Let them adopt, as Mr Stiles had here done, the golden rules of early incision, free drainage, and complete asepsis, and the future would record still better results than the past had done.

*Surgeon-Captain J. Chaytor White* said that he was not the Indian surgeon Dr Leith had referred to. He had had no experience of internal hæmorrhage. The operation they always did now was free opening and free drainage.

*Dr W. Russell* said he would like to say a word before this discussion closed, simply to support what had already been said with reference to the necessity of rather fearless exploration of the liver by means of the aspirating needle. He would almost like to ask *Dr Bramwell* why he delayed so long in exploring this case. The diagnosis, on his own showing, was quite without doubt. He would like to ask *Mr Stiles* why he postponed. When they got a surgeon to see a case in hospital he had for some reason or other a hesitation about putting an aspirating needle into the human body. As a physician he had not had that hesitation, and he could remember one case of this kind, when he was house-physician at Wolverhampton seventeen or eighteen years ago, they aspirated at least six or seven times in the liver without any injury whatever, and without expecting any injury from it. His rule certainly since then had been somewhat fearlessly to work with his aspirator and with its needle, and he must say, in spite of all his admiration for the beauty of the record of this case and for the very excellent result, he was surprised at the hesitancy shown both by *Dr Bramwell* and *Mr Stiles* in the practical application of that most useful instrument.

*Dr Bramwell*, in reply, said that the blood ought to have been examined. Personally he had never seen the malarial organism, though notwithstanding his friend *Dr Laurie's* recent criticism, he certainly believed in its existence. With regard to *Dr Russell's* criticism, he might say that when he first saw the case on October 8, although he thought it was highly probable that there was an abscess, yet he thought it better to try the effect of larger doses of quinine before operating. He did not think that by the delay of a fortnight anything was lost. At the end of that fortnight, as the patient was no better, *Mr Stiles*, who was asked to see the case, with the object of puncturing the liver, thought that, from the surgical point of view, it would be better to wait in order to get some clue as to the position of the abscess. He fully admitted that one could puncture the liver with a clean aspirating needle without any danger; that was acknowledged.

*Mr Stiles* said he desired to thank the Society for the way in which they had received this case. His feeling was that the patient was not losing ground. He was a man of very strong physique, and was able to walk, and a fortnight had not made much difference in the past, and was not likely to. His second reason for delay was this. His teachers had impressed on him the importance of opening abscesses at the time of aspiration. There was no enlargement of the liver, and he had no means of localizing the depth and situation of the abscess. There was always the risk of extensive hæmorrhage. He thought they gained by waiting rather than lost by it.

*Surgeon-Captain White* said that for diagnostic purposes puncture had been done on a certain day, and operation, at most, twenty-four

hours later. It had not been done at time of puncture. Of course, as Dr Bramwell and Mr Stiles said, the diagnosis was undoubtedly obscure in this case. That was a sufficient reason for delaying the puncture.

### 3. GENERAL SECONDARY CARCINOMA OF THE BONES : OSTEOMALACIA CARCINOMATOSA.

By JAMES RITCHIE, M.D., M.R.C.S. Eng., F.R.C.P. Ed., etc. Pathological Report and Microscopical Investigation by JAMES PURVES STEWART, M.A., M.B. Ed., M.R.C.S. Eng.

WE not infrequently meet with cases of primary and of secondary cancer of the bones, and probably if microscopical examination were made post-mortem in every case of secondary cancer it would be found that secondary infection of the bones is not nearly so uncommon as has been hitherto believed. But there are not on record many cases of general secondary cancer of the bones, and still fewer of such general infection associated with softening due to the morbid process, presenting features resembling osteomalacia—*osteomalacia carcinomatosa*. As the following case is an example of this rare condition it is of more than common interest. It is invested with additional interest because of the long time which elapsed between the removal of the primary focus and the manifestation of any symptoms which might be taken as an indication of secondary infection, because of the absence of deposit in the cicatrix and glands, and also because of the microscopical appearances in the bone.

Mrs P., a lady of rheumatic constitution, was in 1878 confined of her second child. After this she enjoyed good health, but on numerous occasions had slight hæmorrhage from the left nipple. This breast continued healthy, but on June 18, 1885, she was seen in consequence of a small lump which she had newly discovered in the substance of the right breast. Two days later Mr Annandale removed the whole of that mamma and some of the axillary glands. The tumour was found to be scirrhus carcinoma. The patient was then aged 47. Recovery was good. The parts were examined at intervals, because if when she suffered from rheumatism the neighbourhood of the right breast was painful she became anxious; the cicatrix and adjacent parts continued healthy, there was no enlargement of glands, and no evidence of recurrence even up to the time of death in November 1894. On February 4, 1892, six years and eight months after the operation, she had an attack of severe pain in the back and limbs. The pain was produced by movement; the muscles of the back were painful on pressure, but in addition there were occasional spasms of pain, darting pain, independently of movement. As this was supposed to be rheumatic the patient was treated in accordance with that

diagnosis. After a few weeks she seemed to be in her usual health. During the attack, although the urine was at first acid it became neutral, with a copious deposit of earthy phosphates, and these conditions remained for a considerable time. While at the coast in summer she suffered from pains and stiffness. In the autumn of 1893 she had much anxiety and fatigue in nursing her husband, who had a hemiplegic attack, and in October she had an illness similar to that of February 1892. From this she never completely recovered, always experiencing a slight amount of discomfort on movement; she was, however, able to go to country quarters during the summer. Early in October 1894, on her return, it was evident that she had lost considerably in stature; she was rather pale, with a debilitated appearance, and stated that during her absence she had suffered from bronchitis and asthma. On examination it was found that both the pelvis and thorax exhibited the changes commonly seen in osteomalacia. The pelvis was beaked, the spine twisted, and the ribs crowded together. The air did not enter the chest very freely, and there were a few sibilant râles. She gradually lost strength, was confined to bed, and on November 25, while being moved in bed, something was heard to crack. It was found on examination that the fourth rib on the right side was fractured. On November 28 she was suddenly seized with dyspnoea; the pulse, which had hitherto been fairly good, became extremely small and feeble. During the last days of life the pressure of the stethoscope on the ribs in front caused slight discomfort. Although death seemed to be imminent, she lingered for four days.

The diagnosis which was made was that the patient suffered from osteomalacia, and that death was precipitated by pulmonary thrombosis. The post-mortem examination was made by Dr Purves Stewart, who submitted the following report upon the pathological condition present:—

The clinical facts of this most interesting case have already been discussed by Dr Ritchie, and I propose to consider the pathological conditions found after death.

Permission was only obtained to examine the thorax, but even this limited examination yielded results which will go far to explain the remarkable clinical phenomena observed during the patient's life by Dr Ritchie.

Dr Ritchie has already referred to the peculiar deformity of the pelvis with its "rostrate" pubic symphysis so strongly suggestive of osteomalacia. He has also told you of the spontaneous fracture of the fourth rib on the right side which occurred some days before death, and I propose to-night to describe the pathological appearances found in the bones, and to offer an explanation why the fracture occurred where it did.



## 1. PATHOLOGICAL REPORT.

The heart was examined *in situ*. The right ventricle was moderately distended, the left ventricle firmly contracted.

The pericardial sac contained about six ounces of straw-coloured fluid.

The right auricle was filled with soft, dark blood-clot which extended through the tricuspid opening into the right ventricle. The tricuspid valve segments were slightly thickened at their free edges. The pulmonary valves were healthy. The pulmonary artery contained a long, firm, pale clot which extended from within the right ventricle as far as to the bifurcation of the pulmonary artery, and was firmly attached to its posterior wall. The left auricle was empty. The mitral valve segments were slightly thickened at their free edges. The left ventricle was contracted and empty. The aortic valves were healthy.

The parietal pericardium was quite healthy, but through it small nodules about the size of sago grains could be seen in the pericardial layers of both pleuræ.

The cellular tissue in the anterior mediastinum was somewhat emphysematous, but I could not trace any direct connexion between the gas in its substance and the lung air.

The right pleural cavity was entirely obliterated by fibrous, somewhat œdematous tissue. The right lung was congested and œdematous, especially in its lower lobes, and showed capillary bronchitis. The convex outer surface of the lung showed opaque nodules about the size of split peas here and there, and these nodules had bands of cicatricial tissue radiating from them along the surface of the lung, causing its surface to be puckered. No nodules could be found in the lung substance.

The bronchial glands were not enlarged, nor were any nodules found in them.

The left pleural sac contained about 30 ounces of blood-stained serum. The left lung was somewhat collapsed. Its parietal pleura was studded with numerous sago-like white nodules, as also was the visceral pleura, the latter more abundantly. The left lung was œdematous, and showed bronchitis, but had no nodules in its substance.

The ribs were all very much thinned, especially close to their sternal ends. The costal cartilages were much thicker than the ribs. There was a fracture of the fourth rib on the right side, quite close to the chondro-costal junction. There was no callus round this fracture, and the rib was so much thinned as to be translucent. So also were all the true ribs at their anterior ends.

I have made microscopic preparations of sections of the ribs, and under the microscopes several sections have been placed illustrating the chief points of interest. I also show some micro-

photographs kindly made for me by Dr E. W. Carlier, and two drawings which I have had made from these photographs. (See Plate I.)

On examining such a transverse section of a rib we at once observe that the bony substance has largely disappeared, and has been reduced in parts to a sheet not thicker, and sometimes even thinner than an egg-shell.

We farther find that this absorption of bone has taken place from within, so that whilst the outer surface of the bone preserves its normal contour, the rib is in reality a very fragile and hollow ghost of its former self.

In the field we can see an army of large cancer cells, arranged in columns or alveoli, with a little interstitial stroma, advancing through the bone marrow,—and the bone marrow in our case seems to play a very passive part; it is apparently quite healthy (Plate II.), and its cells have neither proliferated nor broken down, they are simply pushed aside by the advancing cancer cells.

But when the cancer cells reach the inner surface of the bone much more active pathological reactions are seen to occur. The bone is absorbed, and it is absorbed in two ways:—

(1.) Firstly, by direct action of the cancer cells upon the bone substance. This is well seen in Plate I. (Fig 1, *h*), in which we see a cluster of cancer cells lying in a little bay of bony substance which they have excavated for themselves.

(2.) There is a second way in which the bone is absorbed, and that is by the bone-corpuscles themselves. As the cancer approaches the inner surface of the bone, the bone cells, especially those nearest the advancing cancer, begin to digest the walls of the little bony chambers in which they lie. This is well shown in Plate I. (Fig. 2), in which we see the large cancer cells advancing in columns towards the bone, but separated from it by a little stroma. The cancer cells, therefore, are not yet actually in contact with the bone, but already we can see active changes in the adjacent bone. Some bone-cells are seen to be lying in spaces much too large for them; others are seen to have dissolved away the bone lying between them and the medullary cavity, and they lie at the bottom of tunnels which are open internally towards the medullary cavity of the bone. Finally, we see other bone-corpuscles fully liberated and lying upon the inner surface of the bone. What becomes of these liberated bone-corpuscles I cannot say.

Osteoclasts—the ordinary giant-cells of bone—are conspicuous by their absence.

## 2. SPONTANEOUS FRACTURE OF RIB.

In this case, as Dr Ritchie has described, the signs of fractured rib were quite distinct for several days before death, and yet without the history of any ordinary injury which could be expected to cause such a fracture.

The vast majority of cases of fractured rib are due to violence, and a rib may be fractured either (1) at the point of impact, or (2) about its middle, as when the chest is subjected to a severe crushing force.

In this case, however, the fracture occurred close to the junction of the rib with its costal cartilage. What explanation can be offered to account for this curious fracture?

I believe that the ordinary movements of respiration tended to weaken the fragile cancerous bone at this particular spot, and that the actual fracture occurred either from a sudden deep inspiration or owing to a sudden strain being thrown upon the bone during some movement when the rib was in a state of torsion at this part.

Let us recall a few facts with regard to the physiology of respiration. During respiration the ribs are raised, especially their anterior ends, and they carry upwards and forwards the sternum and its attached cartilages. They are also rotated each around its own long axis, so as to twist the costal cartilages to which they are immovably attached at their anterior ends. This costo-chondral junction is thus exposed to a distinct strain at every inspiration, since the elasticity of the costal cartilages causing them to *untwist* when inspiration is over, is an important factor in ordinary expiration.

The weak point of the rib, then, is where the movable rib joins the relatively immovable costal cartilage by a fixed, unyielding joint.

I have experimented with several dried ribs, so as to imitate the natural movements of respiration,—that is, to keep the anterior end of the rib somewhat fixed, but leaving the costo-vertebral articulation free, so that the rib can be rotated on its own axis. When the ordinary movements of respiration were made with such a rib with sufficient violence the rib fractured, and it fractured always close to its anterior end,—that is, exactly in the situation found in our case.

When we remember the hollow, brittle condition of the bones found in this case, it is not unreasonable to suppose that what powerful muscular movements could perform in a dried rib, ordinary muscular movements can also achieve in a rib already so much weakened by disease.

The foregoing report by Dr Purves Stewart shows that the case was not one of simple osteomalacia, but that it was an example of a more rare disease, viz., general secondary carcinoma of the bones, with softening,—osteomalacia carcinomatosa. As has already been indicated, the features of special interest are, the general distribution of the infection of bones, the association with this of softening and deformity, the long period which elapsed between the removal of the primary focus and the appearance of symptoms

pointing to secondary infection, and the microscopical characters of the bones.

*Frequency.*—Increasing experience points to the conclusion that secondary infection of the bones is much more common than was formerly believed. Hawkins reports cases in which the bones were cancerous, although during life there had been no suspicion of such infection. Arnott records the case of a man, aged 48, who came to him to have a swelling on the temple opened. Two and a half years before, he had his left arm removed for medullary cancer at the upper end of the humerus. He died of influenza. The swelling for which he sought advice was cancerous. There were also other cancerous tumours of the skull; and at the post-mortem it was found that the stump was healthy, but that the clavicle, scapulæ, sternum, vertebræ, ribs, and femur had all disease analogous to that found in the skull, but confined to the cancellous structure. Such infection was unsuspected during life. There was no cancer of internal organs. Förster records a very interesting case, the fourth of his series, that of a woman, aged 49, who died of thrombosis of the right iliac vein, with hæmorrhagic infarcts in several internal organs. The vertebræ and many other bones on section seemed to be healthy, but under the microscope they were seen to be affected by general carcinomatous infiltration. Snow found on post-mortem examination that of ten cases which had been operated on for breast cancer, nine had secondary deposits in the humeri, in four of them the vertebræ were cancerous, and in some of them other bones also were affected. In one case recorded by Snow, a woman who had breast scirrhus, but who died from cerebral and pulmonary complications, the bones were cancerous. A specimen which was taken as characteristic of normal bone marrow was accidentally found to be cancerous. Another patient, who had scirrhus of the mamma of three and a half years' duration, who died of tubercle of the lung, had no symptoms of bone disease, nevertheless scirrhous cells were seen in typical acini in both humeri and in one lumbar vertebra. Snow examined specimens in the museum of St Thomas's Hospital, No. 673, from a woman who had died after excision of the right breast for scirrhus. A section of one femur which had been preserved moist was found to be a typical example of spheroidal-celled carcinoma. The other half of the same bone, the right humerus, and one patella were all mounted as dry specimens of fragilitas ossium; but Snow believes them all to have been cancerous, certainly the femur was.

Förster believed that osteomalacia was a very rare phenomenon in cancerous disease; he had sought for examples both in literature and in museums without success. He had found many observations regarding the softness and flexibility of individual bones. He observed and recorded five cases of carcinoma affecting the

spinal column, thorax, and other bones. Two of these presented a complete picture of osteomalacia, but they were discovered to be cancerous (osteomalacia carcinomatosa) only after post-mortem examination.

The first case was that of a woman, aged 37, who about a year previously had the right mamma and axillary glands removed for carcinoma. The wound healed perfectly with no appearance of recurrence in the cicatrix. She, however, did not regain her strength, grew thinner, the lower half of the sternum increased in size; eventually she died of œdema of the lungs. On commencing the post-mortem examination Förster recognised at a glance the deformities characteristic of osteomalacia,—well-marked kyphosis, alterations in the shape of thorax, etc. On part of the cicatrix there was a thin crust, and in the right axilla a small cancerous nodule. All the bones were affected with diffuse carcinomatous degeneration, and could be cut with a knife or with scissors.

The second case was that of a woman, aged 59, who had been twice operated on because of cancer of the left breast. After the second operation the wound healed well, but the woman continued to lose strength, the spinal column became kyphotic, the left side of the thorax sank in behind and was pushed forward in front; the symphysis pubis projected a little. A secondary nodule developed in the operation scar, and the axillary glands became cancerous. All the vertebræ were cancerous, but the sacrum was free from infection; the intervertebral substance thinner, but not cancerous. Of the ribs a number were affected, showing in some places cancerous infiltration, in others healthy medullary substance.

The third observation related to a preparation in the Würzburg collection, regarding which Förster could not obtain any previous history. The specimen was from an adult male, and consisted of the bones from the tenth thoracic vertebræ downwards with the pelvis. There was kyphosis in such a degree that it must have been observed during life. The vertebræ were infiltrated with scirrhus, so also the spinal cord and membranes, but the pelvis was unchanged.

The fourth case related to a very fine specimen of diffuse carcinoma of the spinal column and many other bones. There was no softening and no deformity. The subject was a woman, aged 49, well built, but emaciated, the right lower limb very œdematous and swollen. The brain and cord were normal; in the left lung a cancer nodule; the pancreas large, hard, with a smooth shining surface on section. Infarcts in several parts of internal organs. An old hard thrombus in the right iliac vein. The bones of the spinal column seemed normal to the naked eye on section; but under the microscope the bodies, processes, and arches showed diffuse carcinomatous infiltration of the medullary substance.

Rokitansky held that osteomalacia occurs not seldom in cancerous disease, and that not infrequently it is associated with cancer of the internal organs. He records the following case:—

A silk weaver, aged 61, as long as he could remember, suffered from rheumatic pains in his limbs. The last year of his life he had very severe, sharp pains in the lower extremities, then in the trunk, especially the thorax. Fever, cough, dyspnoea, and diarrhoea supervened, and he died of marasmus. Post-mortem the body was pallid; the bones of the trunk, the ribs, sternum, and vertebræ were soft and easily indented; they contained a whitish, milky, creamy fluid. The inner surface of the whole vault of the skull was lined with pale, red, lardaceous, medullary (cancerous) adventitious growth.

Volkman said that in primary cancer of the bones there may be deformities of the skeleton resembling in all respects those seen in osteomalacia. He states that in the absence of deformities, and in the absence of cancerous swellings in other organs, the diagnosis, as Förster has pointed out, is often impossible during life. Cases of rapid extension to a great part of the body without deformity are usually mistaken for rheumatism.

Butlin records the case of a man, aged 50, who had softening of all the bones; they could be cut with a knife, and the calvaria bent with the fingers. This was reported to be sarcoma, secondary to two tumours of the lower jaw, one of seven, the other of four years' duration. Butlin remarks that mollities ossium is probably not unconnected with myeloid disease.

Hirschberg records the case of a servant woman, aged 35, who had softening of clavicle, the bones of both arms, left thigh (which was afterwards fractured), left humerus, left tibia. In the bones there were sarcoma, cysts, and softening.

*The Kind of Cancer.*—Förster described his cases as primary, but Volkman was of opinion that all the cases collected by Förster did not relate to primary cancer. Carcinoma has rarely more than one focus of origin; general carcinoma of the bones is therefore secondary. The two most marked of Förster's cases, although described as primary, were secondary to carcinoma of a mamma which had been removed some time previously. A specimen described and figured by Volkman as primary diffuse cancerous infiltration with softening is also secondary to scirrhus of the mamma which had existed for four years. The specimen was from a woman aged 56. Nine months before her death she had pains in the left hip; the limb gradually shortened, and the femur in its upper half bent like caoutchouc. There was also diffuse carcinoma of the lumbar and dorsal vertebræ, of the ribs, and of the left half of the pelvis. The bones were flexible and could easily be cut with a knife. The majority of the cases are secondary to scirrhus, most frequently to breast cancer; indeed, so frequent is the cou-

nexion that Snow stated, in an article in *The Lancet* in 1891, that distant secondary deposits in the bones are always secondary to breast cancer. He had not then seen any which were not, although he recorded references to some. Hawkins, on the other hand, knew of only three cases of scirrhus, as distinguished from medullary cancer, with secondary deposits in the bones. Rindfleisch held that the diffuse carcinosis of the pelvic bones and adjacent viscera, which present the clinical features of osteomalacia, is a soft cancer, characterised by the degeneration of the medulla from a countless number of foci. There are exceptions both in relation to the kind of cancer, and also in relation to the primary seat. Cruveilhier recorded a case in which one of the testes was removed from a young man, aged 27, on account of medullary cancer; six months later he had pain in the sides of the chest and shoulders, and later paralysis of the lower limbs; post-mortem the vertebræ were found to be cancerous, and also the posterior extremities of the first two ribs. Snow also gives references to two examples of carcinoma with uncommon sources of origin,—the specimens in the Museum of the Royal College of Surgeons, England, No. 1687, the parietal bone of a child secondary to carcinoma of the testis, 1687A and 1687B, skull and left femur of a man secondary to carcinoma of the prostate. There are also on record examples of the disease secondary to cancer of internal organs. Paget remarks that the peculiar cancer of the thyroid secondarily affects the bones as if by selection. Moxon records a case of colloid cancer which affected the cranium, spine, seven of the right and four of the left ribs, also one bronchial gland. The patient was a young man, aged 23. The lungs were healthy. The bones of the limbs were not brittle. They were not examined microscopically. Sarcoma occasionally picks out the bones for general infiltration.

The secondary deposits correspond very exactly in character with the primary source of infection, whether it be scirrhous, medullary, or colloid cancer, or sarcoma. It is very evident in melanotic cancer.

*The length of time which may elapse before there is evidence of secondary infection.*—An interesting point in the case recorded is the length of time which elapsed between the operation and the fatal issue, viz., nine years and five months; and if we are warranted in considering the first attack of severe pains in the back and limbs as evidence of infection of the bones, then five years and eight months had passed since the removal of the primary disease. Frederick Dennis says that if there is not evidence of reappearance within three years after operation, there is little probability of it at a later date. Hardly 2 per cent. of cases occur after three years. Snow found that of 44 cases operated upon for breast cancer wherever reappearance had subsequently ensued, it had, with one exception, been obvious within two years, or so short a space of time after-

wards that medical examination would have detected the symptoms after the lapse of two years. The exceptional case was that of a woman operated on in 1879 who seven years afterwards, viz., in June 1886, was readmitted with malignant disease in the liver; she had apparently enjoyed good health in the interval. The cicatrix and the axillary glands remained free from deposit. Holmes records the case of a woman, aged 52, who had scirrhus of the right mamma—a small hard, puckered mass the size of a walnut—for thirty years. She had concealed it through fear of operation: she had suffered pain in the left hip for three years; after that period the bone gave way while being examined by a surgeon; three months later she had fracture of several ribs, and several tubercles in the skin. Post-mortem the ribs were in some places so attenuated that they gave way to the slightest force; numerous soft deposits were also seen in them. Hawkins records the case of a woman, aged 55, whose right breast had been removed six years before for cancer; the part remained well for five and a half years, then tubercles formed around the cicatrix; for two months prior to that she had experienced much pain in the back. Post-mortem all the vertebræ were found to be cancerous and softened; many of the bones, the glands, and the serous membranes were also infected.

In relation to the long interval which occasionally elapses between the primary and the secondary manifestations, it is interesting to notice that the disease has apparently remained dormant for long periods in one or many bones until some accident, or some serious debilitating influence, has served to stir it into activity. The case now recorded is an evidence that in cancer cells the inherent powers of growth are various, apart from the character of the nidus. Bone is said to afford a specially favourable nidus for the development of the cancer cell, and to explain those cases in which the disease remains in abeyance in the bones for a long period, it has been assumed that the *resting spores* of the cancer had been deposited there. The supposed existence of a resting spore has been introduced in order to explain the difficulty presented in these patients. In the present case we have an example of bone infected for so long a time that it had, because of alterations in its nutritive processes, been rendered extremely thin; and nevertheless, when examined, certain portions of the marrow appear to be healthy (Plate II.), they present no evidence of disease, while in the adjacent portions the cancer cells are arranged in characteristic form. The appearance presents a great contrast to those cases in which the whole medullary substance is infiltrated with cancer cells. The reasonable explanation seems to be that the infective cell in this case was one of slow or weak power of development. Apart from the direct influence of the cancer cell on the structures adjacent to it, there must be some interference with the general nutritive changes in



the bone at a distance from the growth. The course is not invariably the same. In some the bones become brittle, but in others they are rendered flexible. From the latter the earthy salts must have been removed to a greater extent than from the former. In the first three cases of Förster, previously referred to, cancerous infection was associated with softening; but in the fourth case, although there was general infiltration of the bone, there was no softening. In some cases the bone is rendered thinner, in others it occupies a larger bulk.

A point of interest in cancer cases is the existence or absence of cachexia. We occasionally meet with patients who have for considerable periods of time suffered from cancerous disease, who appear well nourished, with a good colour; they have none of the cachectic appearance which is exhibited markedly by others, even although the latter are not suffering from septic poisoning. A gradually increasing number of observations point to the interference with the blood-forming function of bone marrow, in consequence of secondary infection, as being at least one of the causes of the anæmia which is seen as a late symptom in some cancer patients. Why is it that sarcoma rarely affects the glands, although it frequently involves the bones? Are the phagocytes of the glands able to deal with the morbid infective material of sarcoma, but is the lymphoid tissue of bone unable to do so because of the difference in the character of its cells? Carcinoma has an extraordinary proclivity for dissemination by the lymphatics, the glands are early affected, and, as in the above case, the bones and serous membranes sometimes become infected while internal organs escape. Why is malignant disease so often confined to a bone or many bones without affecting the adjacent structures and glands, then if the bone is fractured a rapid development succeeds? Is the cause a purely physical one?

*Symptoms.*—The earliest symptom of secondary cancer of the bone is pain in the bone involved, and tenderness on pressure. But there are on record several cases in which the bones were unexpectedly found to be cancerous, there not having been any symptoms of bone disease during life. As the disease advances the nutritive processes in the bone become more seriously affected, and not always in the same way; the function of bone marrow is also interfered with. In some cases the bone becomes brittle, but more frequently it is softened and may be bent, or cut with a knife. In some cases the bone is enlarged; this is most often seen in the sternum. When the function of the bone marrow is affected, and a large number of bones are involved, the blood-making is interfered with, and the patient becomes anæmic; consequently if a patient who has at a former time suffered from primary cancer should become anæmic, apart from septic absorption, then there is a strong presumption that bones have become infected.

*Diagnosis.*—The diagnosis of this, as of other insidious maladies, is difficult. Förster's experience was that he had observed several cases of carcinoma of the spinal column and thorax which presented a complete picture of osteomalacia, and it was only after death discovered to be bone cancer. Volkmann said that in the absence of deformities, and of cancerous swellings in other organs, the diagnosis is often impossible during life. Both osteomalacia and osteomalacia carcinomatosa have been most frequently mistaken for rheumatism. In the early stages of both diseases there is no deformity as a guide; in both of them there may be pains in the bones, aggravated by pressure; the blood-forming function may be interfered with, leading to a degree of anæmia, and although it is said that in osteomalacia carcinomatosa the cachexia is more marked than in the simple disease, it is difficult to differentiate when it is a question of degree of any symptom. Although nerve symptoms may occur in the cancerous variety, they are often absent, whereas in nearly every case of simple osteomalacia they are marked, viz., increased knee-jerk, ankle-clonus, spasmodic twitching. Jaccoud says that osteomalacia carcinomatosa produces rapid painful paraplegia. This is an accidental occurrence, and is uncommon.

If we have to deal with any patient who has suffered from cancer, whether operated on or not, and who is not the subject of septic poisoning, the supervention of anæmia should lead to the suspicion that the bone marrow has become secondarily affected; pain in the bones which is aggravated by pressure should increase the suspicion, and the supervention of deformities in such a case would warrant the diagnosis of osteomalacia carcinomatosa rather than of simple osteomalacia.

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#### LITERATURE.

- ARNOTT.—*Lond. Med. Gazette*, vol. ii., p. 201, 1840.  
 BUTLIN.—*Lond. Path. Trans.*, vol. xxxi., p. 277, 1880.  
 COOK.—*Trans. Path. Soc. Lond.*, vol. xxii., p. 260, 1871.  
 FÖRSTER.—“Ueber die Osteomalacie bei Krebskranken,” *Würzburger med. Zeitschrift*, 1861, Bd. ii., Heft 1.  
 FÖRSTER.—*Ref. Schmidt's Jahrbücher der gesammten Medicin*, ii., p. 17, 1861.  
 HAWKINS.—*Med. Chir. Trans.*, vol. xxiv., p. 45, 1841.  
 HIRSCHBERG.—*Beiträge zur pathol. Anatomie und Physiol.*, Bd. vi., p. 513, 1889.  
 HOLMES.—*Trans. Path. Soc. Lond.*, vol. xi., p. 219, 1860.  
 JACCOUD.—*Traité de Pathologie Interne*, t. ii., p. 556.  
 KOUHN.—*Gazette Hebdomadaire*, p. 729, 1864.  
 MOXON.—*Trans. Path. Soc. Lond.*, vol. xxii., p. 206, 1871.  
 PAGET.—*Surgical Pathology*, 1876, p. 818.  
 PAGET.—*Lancet*, ii. 1887, p. 1002.

RINDFLEISCH.—*Pathological Histology*, New Syd. Soc., vol. ii., p. 293, 1875.

ROKITANSKY.—*Pathological Anatomy*, vol. iii., p. 177.

SNOW.—*Cancers and the Cancer Process*, 1893.

SNOW.—*Reappearance of Cancer*, 1890.

VOLKMANN.—Pitha and Billroth, *Handbuch der Chirurgie*, ii., 1-2, p. 470.

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*Dr William Russell* said he considered this case one of very great interest indeed, and the specimens were very beautiful. The question of bone infection was a very interesting and very important one, and he would regret not to say a few words. This case was important, because it showed clearly and definitely the existence of this condition beyond any question or cavil, and the specimens were so beautifully prepared that no histologist would for a moment doubt that the case was one of cancer of bone. He would like to know if there had been any tenderness, more especially in the ribs. He was much surprised to see that there was no enlargement of bone along with this extraordinary destruction of bone.

*Mr Cathcart* said that he shared *Dr Russell's* great interest in this paper. It had been well worked out both on the histological and clinical sides. He never thought that anybody doubted that there was such a thing as cancer of the bones. Apart from reservations as to its frequency and general dissemination, he thought that had not been recognised sufficiently, judging from what *Dr Ritchie* had told them. In reference to the question *Dr Russell* had raised as to enlargement of bones in this condition, there was one point that had struck him, looking at tumours of bones generally, quite apart from secondary deposits. The conclusion he came to in arranging bone tumours was that, if there was a central tumour growing sufficiently quickly, there would be absorption without enlargement. In those of intermediate rate of growth there was a fibrous capsule. In those growing more slowly there was enlargement and re-formation of bone on the outside of the tumour simulating expansion. In this case the tumour was growing so rapidly as to cause absorption without producing any "expansion" or re-development on the outside.

*The Chairman* referred to a sarcoma he had once exhibited to the Society, occurring primarily in bone, and affecting lungs and liver secondarily.

*Dr Leith* said he could not go into the subject at this late hour, but he had followed the description and statistical history with great interest. The case had been admirably worked up. He could not altogether agree with *Dr Ritchie* in some of the positions he took up. He certainly could not allow that secondary cancer of bone was at all a rarity. In fact, he might almost say it was one of the commonest positions in which secondary cancer

was found. The microscopic preparations showed very well the points which Dr Purves Stewart wished to demonstrate.

*Dr James Ritchie*, in reply, said that what he did say was that, so far from being rare, it was very common to have secondary cancer of bones. The peculiar thing in this case was the general distribution of it. It was not common to find it so generally distributed, he believed, at any rate associated with softening so as to resemble a case of osteomalacia. Another point of great interest was the length of time which had elapsed since the removal of the primary focus. As to the frequency, he thought, with regard to Dr Russell's remarks, that when they got men like Volkmann, Rokitsansky, and Rindfleisch all agreeing as to its frequency, it was not so rare as some men tried to make out. He had had occasion frequently to examine the patient's ribs, and had not found tenderness. There was no evidence of any intense congestion or proliferation to be seen in the microscopic sections. He believed that tenderness on pressure and pain in bones were associated commonly with congestion of the periosteum, and in this case they had no evidence whatever of any inflammatory action. This might explain the absence of tenderness. So far from there being enlargement of bones, they were smaller.

# APPENDIX I.

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## ON SEPARATE ACROMION PROCESS,

WITH

## APPENDIX ON SUB-CORACOID DISLOCATION OF THE HUMERUS.

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## ON SEPARATE ACROMION PROCESS SIMULATING FRACTURE.

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HAVING met with this condition frequently in the dissecting-room I was led to examine the exact anatomy of the part and to consider the causes that might bring about and maintain the condition. Most surgical museums probably contain one or more specimens of this condition, marked as cases of "ununited fracture." When we look at the exposed position of the acromion, and its thinness in front of the beam that rises to it from the spine, we would expect fracture of the acromion to be a more frequent occurrence than the statements in surgical text-books imply. Evidence that true fracture of the acromion, occurring at various places and in various directions, is not infrequent, but is overlooked from the absence of displacement, will fall to be noticed below in order to draw the distinction between the appearances in that occurrence and those in the condition I have to describe.

The condition in question may, possibly, still come within the category of fracture in the sense that it may, in some cases, have begun as a fracture of the layer of cartilage between the basi-acromion and the ossified epiphysis; and, in that event, the movements of the acromion against the clavicle might be sufficient to prevent union and to establish a joint between the two parts of the acromion. That, however, is not likely to have been the history in cases (as in Case No. 3, figs. 6 and 6a) in which the separation exists on both sides.

The relation of the clavicle to the acromion is a fundamental consideration in the inquiry, whether as regards true fracture or the condition in question. The normal anatomy, as bearing on that relation, has first to be considered; then the development of the acromion; and thereafter we are in a position to appreciate the distinction between the condition seen in the specimens which I have to describe and the condition of true fracture of the acromion.

The condition of separate acromion process, in whatever way arising, is not without practical interest. Although, in a case of supposed fracture, the time for crepitus may be past, the condition may be recognisable in the living body by the amount of motion; and, if noticed some time after a contusion of the shoulder, a question might arise whether the contusion had been the cause of the alleged fracture. Again, the not infrequent association of the condition of separate epi-acromion with the condition of the shoulder joint, usually called "rheumatoid arthritis," referred to below in connexion with Case No. 1, is of interest in its bearing on the question of the usual causation of the latter as well as of the former of these conditions.

### I. PRELIMINARY ANATOMICAL CONSIDERATIONS.

*Relation of clavicle to acromion. Ligaments and movements. Interior of acromio-clavicular joint. Adaptations. Inter-articular fibro-cartilage. Various forms of Acromion Process.*

(a.) The *relation of the clavicle to the acromion* is an important consideration in looking for a natural cause of continued separation of the epiphysis of the acromion. The natural relation of the parts is seen in fig. 1. In the abutting of the acromion against the end of the clavicle during the movements of the scapula there is considerable transverse force. Shock is lessened by various adaptations; the very oblique direction, outwards and backwards, of the clavicle; and the form of the joint, which allows of movement in any direction.

(b.) *Ligaments and movements.*—The joint is in itself a weak one, serving merely as a pivot on which the scapula moves. Its great securing ligament is the distant trapezoid part of the coraco-clavicular ligament. The superior acromio-clavicular ligament is a fairly thick strap, about an inch in breadth and in length, and is continued round the front and back, and from the front a little way in below. The so-called inferior acromio-clavicular ligament beyond that is a very weak structure, at the middle only a thin strengthening of the synovial capsule, scarcely worthy of being called a ligament. It is a part very seldom examined in the dissecting-room.

These parts of the ligament afford some check to over-movement; the anterior and posterior parts to forward and backward gliding of the acromion; the superior part to vertical gliding; and the several parts come variously into play in checking rotatory movements of the scapula on the clavicle, that is, movements in which the lower angle of the scapula moves forwards or backwards. The superior ligament is generally regarded as a provision to prevent the clavicle slipping over the acromion (more correctly expressed, the acromion slipping under the clavicle, as the scapula is the moving bone), but it is not evident how a ligament placed



above will check such a movement more than one placed below would. The function of a ligament is seen by looking at the direction of its bundles. Those of this ligament are directed outwards and forwards, as seen in fig. 1, continuing the direction of the outer end of the clavicle, and the adaptation of the ligament seems to be to resist outward traction, as by the weight of the scapula and limb, or by the traction of the deltoid muscle when its humeral attachment is the more fixed point. As above remarked, the coraco-clavicular ligament is really the great ligament of the acromio-clavicular joint, and the scapula is further naturally held up by the trapezius muscle.

(c.) *Interior of the acromio-clavicular joint.*—The appearances may be here noted as the condition of this joint has to be observed in cases of separate epi-acromion. Very frequently, and apparently independent of disease or age, the articular cartilage *on the clavicle* is not smooth and polished like ordinary articular cartilage, but in a spongy condition,  $\frac{1}{12}$  to  $\frac{1}{10}$  inch thick, giving it the character of a cushion. The *facet*, as seen on macerated bones, is often irregular and foraminated on the surface, and indistinctly bordered. Normally the facet occupies the whole of the true outer end of the bone in length and depth. The end may be sharply square-cut, but behind the articular surface proper the border of the bone rounds off backwards and inwards, broadening the bone, so that the seeming posterior angle may be  $\frac{1}{2}$  inch or more from the true end. This rounded part is the rough ligamentous area for the attachment of the back part of the superior ligament. In cases of separate epi-acromion the posterior bundles of this part of the ligament attach the clavicle to the basi-acromion, and the deeper bundles separate the acromio-clavicular synovial cavity from that of the intra-acromial joint.

The *facet on the acromion* is generally better defined, the cartilage smooth; and, on the macerated bones, the articular lamina less foraminated. Whether it reaches to the front of this border of the acromion depends on the form of the latter, as seen in the different figures given. Care is required in defining the posterior end of the facet, a point of interest in deciding whether, in cases of separate epi-acromion, the clavicle has rested in part on the basi-acromion. Behind the facet proper there is a small triangular area,  $\frac{1}{4}$  to  $\frac{1}{2}$  inch in length, the apex meeting the anterior apex of the bevelled impression on the spine of the scapula for the attachment of the trapezius muscle. The upper part of this area attaches the deeper part of the superior acromio-clavicular ligament, the remainder may be foraminated or smooth. The fore part of this area is apt to be taken for the back part of the facet, but the limit of the facet proper is marked by a finished line. In some of the cases of separate epi-acromion noted below (Nos. 6 and 10, and shown in fig. 7), we have to see that the facet does reach for a small way on the basi-acromion.

Normally the facet has an average length of  $\frac{2}{3}$  inch ; depth about half the length ; form elliptical, lower edge generally the least bent ; surface nearly plane, but usually a little concave both ways. The end of the clavicle has its curves the reverse, but it is often irregular.

(d.) *Adaptations of the joint.*—In direction, antero-posteriorly, the acromio-clavicular joint is nearly at right angles to the axis of the outer part of the clavicle, the direction of which is outwards and a little forwards, and the line of the joint is directly intersected by the line of the bundles of the superior ligament. The clavicle thus, in the antero-posterior direction, abuts directly against the acromion. Vertically, the oblique cutting, downwards and inwards, is, in some of the cases of separate epi-acromion noted below, very little, but is usually marked.

This oblique cutting is supposed to be an adaptation to prevent the displacement upwards of the scapula during force from below. The adaptation would be more correctly expressed by saying that, as the plane of the acromion is oblique downwards and outwards, the slant at the joint brings the acromion more directly against the clavicle, thereby giving it a better pivot of support to move on, than if the intersection had been vertical. The obliquity of the intersection is, however, generally greater than is required to make it rectangular to the acromial plane, and will, so far, correct the tendency there would have been for the acromion to be displaced upwards if the intersection had been vertical. Antero-posterior gliding at the joint will tend to obviate shock more than the vertical gliding will. Both are soon checked by the ligaments, mainly by the coraco-clavicular.

(e.) The *inter-articular fibro-cartilage*, partial or complete, that used to be described at the acromio-clavicular articulation as a further provision for obviating shock, I find to be rarely present. Though I have often looked for it, I have only once in my dissecting-room experience met with a fibro-cartilage here with complete synovial cavity on each side. That was in Case No. 4, noted below. Nor does a partial wedge-shaped fibro-cartilage occur often, such as that noted below in Case No. 3, best marked on the right side. There are often, indeed generally, synovial fringes projecting into the joint, especially hanging into it from the superior ligament, which probably have been taken for a partial fibro-cartilage. But when the latter occurs its cartilaginous nature is evident.

(f.) *Various forms of the Acromion Process.*—The general form of the acromion in cases of separate epi-acromion appears frequently to be unusual. Irrespective of that condition, the acromion varies much in form, but, for description, four types may be defined, between which there are intermediate forms. Figs. 10 to 13 show these four types diagrammatically, arranged in the order of frequency. The equal frames, with equal subdivisions, enclosing or intersecting the figures, will assist the eye in recognising the

parts that are wanting or excessive. The facet for the clavicle is indicated on the inner side of each.

(1.) *Quadrangle form* (as in fig. 10); posterior angle prominent, may approach to nearly a right angle, but generally somewhat rounded. Anterior end square-cut, presenting antero-external and antero-internal angle; in some so sharp-cut here as to make two nearly right angles; not projecting beyond clavicle, but continuing the line of curvature of anterior border of clavicle onwards to the antero-external angle, the most projecting part of the acromion anteriorly. Outer border moderately convex, undulating if the tubercles are well marked. Inner border, behind facet, short, and forms either obtuse angle or concavity with upper border of crest of spine.

(2.) *Ovoid form* (as in fig. 11); posterior angle may be more rounded, but chief character is the blunt-pointed projection of anterior end beyond clavicle. The facet thus does not reach to fore part of inner border.

(3.) *Triangular form* (as in fig. 12); antero-external angle deficient.

(4.) *Crescentic form* (as in fig. 13); posterior angle and antero-external angle both deficient. In the latter two types the outer border of the acromion is much bent.

The outer edge of the acromion generally shows a series of tubercles with intervening smooth depressions, seen on the upper aspect, corresponding to the attachment of the intra-muscular tendinous septa of the deltoid muscle. The tubercles, besides the projection of the point and that of the posterior angle, are usually three in number. The tubercles are more easily reckoned than the spaces. It is noted below with each case how many of these tubercles are carried by the separate epi-acromion.

## II. DEVELOPMENT OF THE ACROMION.

Fully the larger part of the adult acromion is formed by ossification from the spine. That is seen in the figures 2, 3, 4, and 5. In all my specimens that ossification forms the posterior angle of the acromion. Towards it the cartilaginous acromion, already with its definite shape in the young subject, sends a narrow tail-like process backwards, but not reaching quite to the angle. The line of junction of the ossifying basi-acromion with the cartilaginous epi-acromion is convex, generally most prominent to the inside of the middle, and slopes very obliquely outwards and backwards to near the posterior angle. The ossifying basi-acromion shows a series of projections or tubercles, like the knuckles of the closed hand, generally seven in number, sometimes pretty uniformly arranged (as in figs. 2 and 5), sometimes with irregularities in size (as in figs. 3 and 4). The irregularity seen in fig. 4 is in adaptation to the osseous centres of the epi-acromion; that seen in fig. 3 is before ossific centres have appeared in the epi-acromion.

*Notes of five specimens showing stages of ossification of the  
Epi-acromion.*

The following notes, and the figures referred to, show the stages of ossification of the acromion process, as seen in the series of specimens of young scapulæ in my collection. As I am unable to give the ages, the dimensions of the scapulæ and the condition of each in regard to the ossification of its other parts are given, but the progressive steps of the ossification are seen.<sup>1</sup> The notes of each should be read in connexion with the figures (figs. 2, 3, 4, and 5). The following Table (Table I.) will facilitate comparison between the five young specimens noted.

Table I.—*For comparison among the five specimens of young scapulæ noted below, relating to ossification of the epi-acromion; the measurements of the scapulæ given in inches, those of the epi-acromion in  $\frac{1}{16}$ ths of an inch.*

Number of Specimen.	Figure.	Scapula.		Entire Acromion.		Length of part formed by Epi-acromion.	Condition of Epi-acromion.
		Length.	Breadth.	Length.	Breadth.		
1	2	3 $\frac{1}{4}$	2	1 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{16}$	Cartilaginous.
2	3	4 $\frac{1}{4}$	2 $\frac{3}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{16}$	Ossific centre at point.
3	4	5 $\frac{1}{8}$	3 $\frac{3}{8}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{16}$	Seven ossific centres.
4	...	4 $\frac{5}{8}$	3 $\frac{5}{8}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{16}$	Ossification advanced.
5	5	5 $\frac{3}{8}$	3 $\frac{5}{8}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{16}$	Epiphysis completely ossified.

<sup>1</sup> *Usual order and periods of ossification.* The following are the ages assigned for the appearance of ossific centres, and for the consolidation of the several parts of the scapula, in the latest edition of *Quain's Elements of Anatomy*, now appearing, 10th ed., vol. ii., part 1, 1890. Centre for the body, 7th or 8th week intra-uterine, spine showing about 3rd month; centre for coracoid in first year after birth; for the sub-coracoid ossicle, at top of glenoid cavity, about 10th year; coracoid joins body about age of puberty; centre in cartilage of base, about 16th to 18th year, beginning at lower angle and thereafter extending upwards along base. "A thin lamina, in two pieces, is also added along the upper surface of the coracoid process, and another at the margin of the glenoid cavity. These epiphyses are united about the 25th year." Of the *acromion* the account is:—"In the acromion two, sometimes three, nuclei appear between the 14th and 16th years; they soon coalesce, and the resulting epiphysis is united to the spine from the 22nd to the 25th year." Of three figures given, one, at 15 or 16 years, shows one nucleus at about the centre of the cartilage; one, at 17 or 18 years, shows two nuclei, one in front of the other along the middle; the third shows the completed epiphysis. In the latter, the line of meeting with the basi-acromion corresponds generally to what is seen in my specimens, but the prolongation of the epiphysis backwards is too broad and goes back so as to include the posterior angle. The condition shown in the first two figures is quite unlike what is seen in my specimens and shown in my figures.

*Specimen 1.* Young scapula in which the epi-acromion is still entirely cartilaginous. Fig. 2.

Length of ossified scapula  $3\frac{1}{4}$  inches, breadth 2. *Acromion*, general form quadrate, posterior angle somewhat rounded; length  $\frac{1}{2}$  inch, of which ossified basi-acromion forms  $\frac{1}{2}$ . *Basi-acromion*, breadth rather greater than length; the ossification continued from spine shows seven rounded tubercles projecting like knuckles of closed hand, in a line falling away a little to inside but mainly curving downwards and outwards, the last one broad and forming the posterior angle of the acromion. *Epi-acromion*, as yet without ossific centre; sends a narrow tail-like strip backwards to near, but not quite to, the ossified posterior angle of the acromion.

*Specimen 2.* Scapula larger than the preceding; one ossific centre in epi-acromion. Fig. 3.

*Scapula*, length  $4\frac{1}{2}$  inches, breadth  $2\frac{3}{4}$ . *Acromion*, quadrate; length  $1\frac{1}{2}$ , of which ossified part forms  $\frac{7}{8}$ , breadth of latter  $\frac{8}{8}$ . Line of the tubercles of basi-acromion irregular (as seen in fig. 3), not turning backwards till on outer third. *Epi-acromion*, only one small ossific centre, occurring at anterior end towards antero-internal angle. The caudate strip of cartilage goes back to near the posterior angle, meeting the last tubercle of basi-acromion in front of the ossified posterior angle.

*Specimen 3.* Larger scapula than the preceding; several centres of ossification in epi-acromion. Fig. 4.

*Scapula*, length  $5\frac{3}{8}$  inches, breadth  $3\frac{3}{8}$ , small cartilage at lower angle not yet ossifying; vertebral border below spine concave; coraco-scapular suture beginning to disappear but mostly visible; scale-like epiphysis on coracoid ossified. This epiphysis corresponds to attachment of coraco-clavicular ligament; coraco-scapular suture crosses under its back part. *Acromion*, form triangular with convex outer border; length  $1\frac{9}{8}$ , of which ossified part forms 1 inch; breadth  $\frac{9}{8}$ . *Epi-acromion*, ossific centres numerous, seven in all (see fig. 4); (a) one at the point, small; (b) two on the inner side, one of them small, the other long and resting on the two inner tubercles of basi-acromion; (c) three on outer side, two of them small, one of them long, the largest of the whole, resting in hollow between two tubercles of basi-acromion. The greater part of the breadth of the epi-acromion, that between the outer and inner series of ossific centres, is not ossified. (d) A centre placed in the position of the caudal process, in front of posterior angle of acromion. This accessory epiphysial centre ( $\frac{6}{8}$  inch in length,  $\frac{1}{2}$  in breadth), is seen very distinctly on the under surface and as forming the edge of the acromion here, but is narrowly seen on the upper aspect, not so broadly as put in the figure (fig. 4) in order to show its position longitudinally. It is at a distance of  $\frac{2}{8}$  inch from the long external centre in front of it. The strip of cartilage that no doubt contained this ossific centre has not been preserved in the specimen. In specimen No. 5 we have to see this accessory centre joined to the one in front of it by a narrow neck.

On the outer margin of the acromion, three moderate prominences are seen; one behind, at the accessory centre; two in front of that, one related apparently to the position of the greater ossification, the other to the position of the two lesser ossifications.

*Specimen 4.* Smaller scapula than the preceding (No. 3), but in which ossification of the epi-acromion is much more advanced (not figured).

*Scapula*, length  $4\frac{5}{8}$  inches, breadth  $3\frac{2}{8}$ ; small cartilage at lower angle, as in preceding case, not ossifying. Vertebral border below spine, convex. Coraco-scapular suture obliterated except on subscapular aspect; scale-like epiphysis on coracoid well ossified. *Acromion*, of the marked triangular type, length  $1\frac{9}{16}$ , of which ossified acromion forms 1 inch; breadth  $\frac{1}{2}$ . *Epi-acromion*, anterior and middle part entirely ossified, but posterior caudate part lost, exposing the two outer tubercles of the basi-acromion and part of the next tubercle. While the ossification of the epi-acromion is continuous, thicker parts are seen; one on the outer border, one at the point, and one along the inner border, the latter bearing the whole of the clavicular facet. These thicker parts correspond to the position of the centres of ossification noted in the preceding case (No. 3), and are separated by depressed parts with a finely pitted surface, the thicker parts smooth, the inner thickening least so. These thickenings are less marked on the under surface, nearly the whole of which is finely pitted. The ossified epi-acromion has a yellow colour, which, with the mottling on the surfaces, reminds one of the appearance presented by calcified sternal ribs, contrasting with the even surface of the basi-acromion.

In regard to the probable age of this scapula, it corresponds pretty nearly in size to one known to be at the 17th year, female, from which the epi-acromion is lost. Coraco-scapular suture seen all round.

*Specimen 5.* Scapula with epi-acromion completely ossified but not united to the basi-acromion; showing distinctly the relation of ossification to the formation of permanently separate epi-acromion. Fig. 5.

*Scapula*, length  $5\frac{3}{8}$  inches, breadth  $3\frac{5}{8}$ ; small epiphysis ossifying on base close to lower angle; base below spine, convex; coraco-scapular suture has entirely disappeared; scale-like epiphysis on angle of coracoid well seen and still separate. *Acromion*, of mixed type, broadly crescentic behind and at middle, but antero-external angle prolonged; length of entire acromion  $1\frac{1}{2}$  inches, of which epi-acromion occupies the  $\frac{1}{2}$ ; breadth at middle  $\frac{1}{2}$ .

The appearances presented by the now ossified *epi-acromion* deserve particular mention. As noted with the preceding specimen (No. 4), there are thicker parts along the sides and at the point. The external thickening is the most marked; extends along the anterior  $\frac{3}{4}$  of the acromion to the point. The part at the point ( $\frac{1}{2}$  inch in length and broadened) is marked off from the latter by a depression, and from the internal thickening by a recess at the middle of the very obliquely-cut anterior end of the acromion, as shown in the figure (fig. 4). The thickening on the inner side is less marked, but is distinct and carries the whole of the clavicular facet. The external thickening is all along marked by an inner sharp serrated edge, which at the middle of the acromion curves inwards as far as to reach the middle tubercle of the basi-acromion. On the under aspect, the distinction of the thickenings is less marked; the surface coarsely pitted, except on the back part of the outer thickening.

The posterior *caudate* part of the epi-acromion is well seen here. Joining the narrow posterior end of the main part of the epi-acromion by a narrow neck ( $\frac{3}{8}$  inch in length,  $\frac{1}{8}$  in breadth), it broadens backwards as an ovoid scale ( $\frac{1}{2}$  in length,  $\frac{1}{8}$  in breadth) as shown in the figure (fig. 5). It is seen on the upper aspect and on the outer border of the acromion, and forms half the thickness of the outer border, but is barely seen on the under aspect. The posterior end of this caudate process does not go so far as the posterior end of the outer border of the acromion, the angle being, as usual, ossified from the spine. Along the outer border of the acromion, three moderate elevations are seen besides that formed by the point, and the one formed by the caudate process behind its narrow neck, at which a concavity is seen on the border.



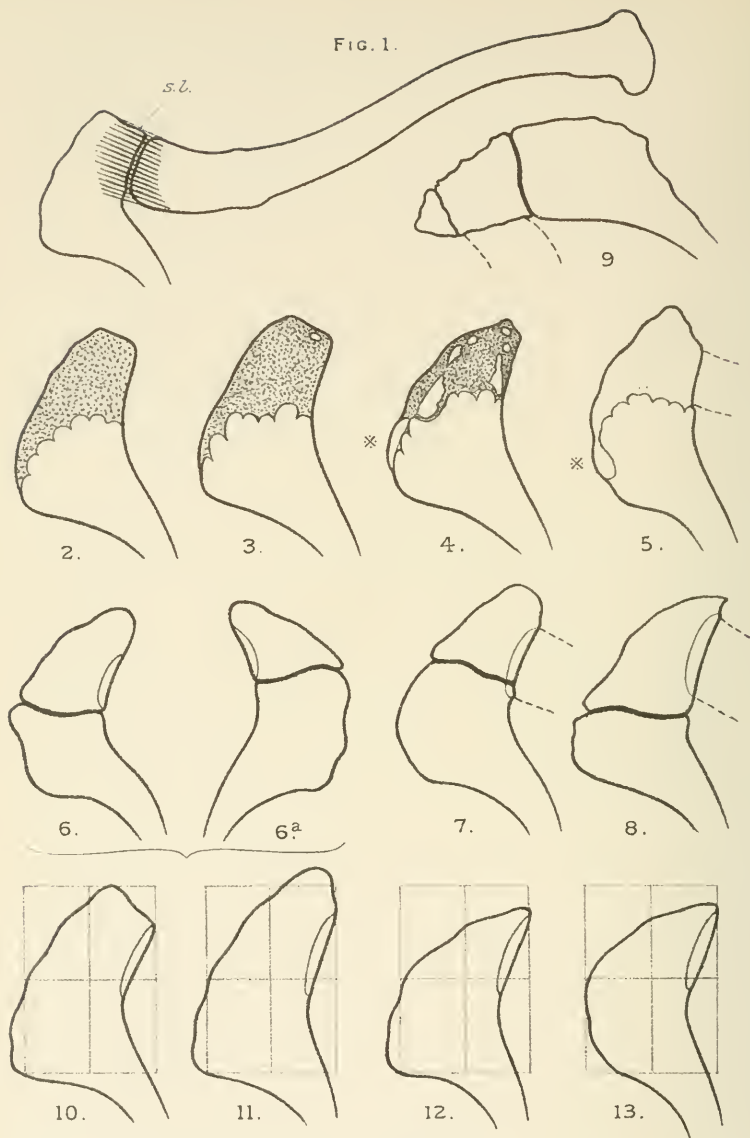


Fig. 1, Normal anatomy. Reduced  $\frac{1}{2}$ .

Figs 2 to 5, stages of development.

Figs 6 to 9, cases of separate epi-acromion in adult or old.

Figs 2 to 9, variously reduced to same size, for comparison.

Figs 10 to 13, diagrams of typical forms.



These particulars in regard to the ossification of the acromion appear worthy of notice in relation to the adaptations of the acromion, and to the occasional occurrence of permanently ununited epi-acromion. We see the earlier ossification of the epi-acromion at its outer and inner sides than at the middle; at the outer, in relation to the attachment of the deltoid muscle; at the inner, in relation to the support of the clavicle. The accessory centre noted with specimen No. 3, is seen in specimen No. 5 to unite by a narrow neck with the main part of the epiphysis, forming a kind of splint between the two. Should this union fail to take place, we have a precise explanation, if not of the cause of permanently ununited epi-acromion, at least of the form which it presents.

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#### EXPLANATION OF THE PLATE.

FIG. 1.—Normal, from a preparation, reduced  $\frac{1}{2}$ . Shows oblique direction of clavicle and relation of clavicle to acromion; *s*, *l*, superior acromio-clavicular ligament, direction of its bundles in direction of outer curve of clavicle; acromion of quadrate type; on outer border the tubercles with intervening depressions.

FIGS. 2 to 4.—Show ossification of basi-acromion from the spine, with knuckle-like projections; and stages of ossification of epi-acromion. In figs. 2, 3, and 4 the dotted parts are cartilage; fig. 2 entirely cartilaginous; in figs. 3 and 4 the ossific centres are white; in fig. 3, one small ossific centre towards anterior end. In fig. 4, seven centres; one anterior; two internal, at clavicular connexion; three external; one posterior, in caudate process of the cartilage, marked with \*.

FIG. 5, ossification of epi-acromion completed, but not yet united to basi-acromion; thinner middle area between inner and outer thicker parts; thick outer part bounded by sharp line internally; posterior nucleus, \*, now united to main part of epi-acromion by narrow neck, non-ossification of which would accord with form in permanently separate epi-acromion.

FIGS. 6 and 6*a*, Case 3 of dissection of soft parts; separate epi-acromion on both sides; female *æ*t. 82; left epi-acromion ossicle larger than right, but both seen to carry entire clavicular articulation; clavicular facet indicated (here and in the following figures) by line marking off a narrow crescentic area. Form of acromion between ovoid and crescentic types.

FIG. 7.—Case 6. This figure given to show case in which ossicle is shorter than usual, in proportion to basi-acromion; acromion mostly of ovoid type.

FIG. 8.—Case 7. Given to show the ossicle longer than usual, in proportion to basi-acromion; acromion of the triangular type.

FIG. 9.—Case 11. Acromion of right side; this figure given to show two epi-acromial ossicles, point of the epiphysis remaining separate from the main part; some traces of disease on this specimen.

FIGS. 10 to 13.—Diagrams to show typical forms presented by the acromion process, placed in order of frequency; equal frames, with equal subdivisions, drawn to assist eye in recognising the different forms. Fig. 10, quadrate form; fig. 11, ovoid form, anterior end prolonged without angles; fig. 12, triangular type, antero-external angle deficient; fig. 13, crescentic type, posterior and antero-external angles both deficient.

### III. ACCOUNT OF DISSECTIONS AND SPECIMENS SHOWING SEPARATE ACROMION OSSICLE IN THE ADULT.

*Points to be attended to in the study of such specimens.*—Careful study of the parts is essential in such an inquiry. Attention has to be given to the following:—Whether there is evidence of disease at the acromion or of the shoulder joint, or evidence of former injury. Sex, age, and robustness of the scapula. General form of the acromion and its size. In regard to the separate *ossicle*,—its length in proportion to the basi-acromion. In regard to the *intra-acromial joint*,—its direction, in relation to the longitudinal axis of the acromion; direction particularly at the outer side, whether obliquely backwards; ligaments and edges or crests at the joint, and amount of movement allowed; nature of the surfaces of the joint, whether a synovial cavity present. In regard to the *acromio-clavicular joint*,—whether the clavicle rests in part on the basi-acromion; whether the ligaments and surfaces are normal.

The following Table may be referred to for comparison of the specimens.

Table II.—*Showing the size of the separate acromial ossicle in relation to that of the entire acromion, and other particulars of each of the scapulae; the measurements of the scapulae given in inches, those of the ossicle in  $\frac{1}{16}$ ths of an inch.*

No.	Fig.	Side.	Age.	Sex.	Scapula.		Entire Acromion.		Separate Ossicle.	
					Length.	Breadth.	Length.	Breadth.	Length.	Breadth at Base.
1	.	R.	80	?	5 $\frac{1}{2}$	4	1 $\frac{9}{16}$	1	1 $\frac{9}{16}$	1 $\frac{9}{16}$
2	.	L.	80	F.	5 $\frac{1}{2}$	3 $\frac{7}{8}$	1 $\frac{11}{16}$	?	1 $\frac{9}{16}$	1 $\frac{11}{16}$
3	6a	R.	82	F.	5 $\frac{1}{2}$	3 $\frac{7}{8}$	1 $\frac{11}{16}$	1 $\frac{11}{16}$	1 $\frac{9}{16}$	1 $\frac{11}{16}$
"	6	L.	"	"	same	same	same	1 $\frac{11}{16}$	1	1 $\frac{9}{16}$
4	.	L.	80	F.	5 $\frac{1}{2}$	3 $\frac{7}{8}$	1 $\frac{9}{16}$	1 $\frac{11}{16}$	1 $\frac{9}{16}$	1 $\frac{9}{16}$
5	.	R.	64	M.	6 $\frac{1}{2}$	4 $\frac{1}{2}$	2	1 $\frac{11}{16}$	1 $\frac{11}{16}$	1
"	7	L.	"	"	6	same	1 $\frac{11}{16}$	same	1 $\frac{9}{16}$	1 $\frac{11}{16}$
6	7	L.	"	?	6	3 $\frac{7}{8}$	1 $\frac{11}{16}$	1	1 $\frac{9}{16}$	1 $\frac{11}{16}$
7	8	L.	"	M.?	6 $\frac{5}{8}$	4	2	1 $\frac{2}{16}$	1 $\frac{5}{16}$	1
8	.	L.	"	"	5 $\frac{5}{8}$	4	1 $\frac{5}{16}$	1	1 $\frac{5}{16}$	1
9	.	L.	"	?	6	3 $\frac{1}{2}$	1 $\frac{5}{16}$	1	1	1
10	.	L.	"	?	6 $\frac{1}{2}$	3 $\frac{7}{8}$	1 $\frac{5}{16}$	1	1 $\frac{5}{16}$	1 $\frac{9}{16}$
11	9	R.	"	M.?	.	.	2 $\frac{9}{16}$	1 $\frac{3}{16}$	{ 1 $\frac{5}{16}$	{ 1 $\frac{5}{16}$
12	.	L.	"	M.?	5 $\frac{1}{2}$	4	1 $\frac{5}{16}$	1	1 $\frac{5}{16}$	1
13	.	R.	"	M.?	6 $\frac{1}{2}$	4	2	1 $\frac{2}{16}$	1 $\frac{2}{16}$	1

*Explanation of the measurements, etc., in the Table.*—*Scapula*, length taken between upper and lower angles; breadth taken along base of spine, vertebral border to posterior border of glenoid cavity. *Acromion*, including the ossicle, length taken along middle; breadth taken at middle of basi-acromion, generally the greatest breadth. *Ossicle*, length taken from middle of tip to middle of its joint with basi-acromion; breadth taken along base at joint, whether transverse or oblique. All the measurements are taken with callipers. As the acromion, as a whole, is bent longitudinally, convexity upwards, there falls to

be added for the length of the basi-acromion about  $\frac{1}{12}$  inch more than that remaining after deducting the length of the ossicle.

The first five cases are dissections, so that the age and sex could be given. In Nos. 3 and 5 both sides are given. The other side of No. 2 was noted as normal. In No. 11 there are two ossicles, anterior and posterior, with slight marks of disease. In No. 2 the acromion was thinned by absorption. In No. 1 there had been sub-coracoid dislocation and rheumatoid disease of the shoulder joint. The remaining eight are macerated specimens. Those that are probably male are marked M. ?; those of which the sex is quite uncertain have ? in the sex column.

#### (A.) DISSECTIONS.

*Case No. 1.* Also old-standing sub-coracoid dislocation of humerus, with chronic rheumatoid arthritis of shoulder joint and great alteration of the anatomical neck of the humerus. From a subject æt. 80, sex not noted. Right scapula, left not noted.

*Scapula* rather undersized (length  $5\frac{1}{2}$  inches, breadth 4) but muscular markings pretty well developed. *Acromion*, of the broadly crescentic type; much bent, very convex at middle third at and behind intra-acromial joint; but a posterior angle present where spine meets acromion considerably internal to usual position of posterior angle. *Ossicle*, triangular, end blunt, like end of little finger, and does not project beyond clavicle. Length  $\frac{19}{32}$  inch, breadth at base  $\frac{19}{32}$ . Entire acromion, length  $1\frac{9}{32}$ , breadth at middle of basi-acromion 1 inch. The ossicle shows one faint projection on outer margin and projects considerably at base. Basi-acromion shows two tubercles besides the posterior angle, one of them at the joint.

*Intra-acromial joint*, direction nearly transverse, undulating, has small median projection and goes backwards at both sides, most at the outer side, giving moderate backward direction to joint externally. Good superior and inferior ligaments, superior the strongest; moderate ridges above and below attach the ligaments. Movement distinct. When joint opened, synovial cavity seen throughout; surfaces ( $\frac{19}{32} \times \frac{5}{12}$  inch) undulating, smooth but finely dimpled, not like ordinary articular cartilage except on most of inner half; covering of cartilage thin. The ossicle is thicker ( $\frac{5}{12}$ ) than the basi-acromion ( $\frac{4}{12}$ ) and rough below, but not diseased.

*Acromio-clavicular joint*.—Inferior ligament considerably strengthened; no inter-articular fibro-cartilage and but few synovial fringes. Cartilage on clavicle thick and mostly spongy, giving irregular surface. Acromial facet, smooth cartilage; height  $\frac{4}{12}$  with the usual inclination; length  $\frac{8}{12}$ , of that  $\frac{2}{12}$  on basi-acromion. When clavicle pushed outwards, ossicle is moved freely; when clavicle pushed outwards and backwards, the movement is arrested by the basi-acromion. Synovial cavity appears to be continuous with that of intra-acromial joint, but I am not certain of that. Coraco-clavicular band strong, attached entirely to the ossicle.<sup>1</sup>

*Case 2.*—With great thinning of acromion; shoulder joint healthy. The appearances suggest former disease of the sub-acromial bursa. From a female subject æt. 80. Left scapula, right scapula noted as normal.

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*Scapula*, rather undersized (length  $5\frac{2}{3}$ , breadth  $3\frac{2}{3}$ ), very thin, very translucent at both fossæ; ridges on subscapular fossa well marked. Surfaces of shoulder joint, capsular ligament, and tendon of biceps healthy. *Acromion*,

<sup>1</sup> The condition of the shoulder joint in this case is noted in the Appendix.

may have been of quadrate type, but now has on outer border three irregular excavations with serrated edges, giving two triangular peaks between. The anterior excavation ( $\frac{6}{12}$  wide,  $\frac{1}{12}$  deep) is entirely on the ossicle; the middle excavation (1 inch wide,  $\frac{3}{12}$  deep) has its apex at the intra-acromial joint and is more on the ossicle than on the basi-acromion; the posterior excavation ( $\frac{9}{12}$  wide,  $\frac{2}{12}$  deep) is on the back part of the outer border of the basi-acromion, the peak bounding it in front is opposite the middle line of the spine of the scapula. The acromion as a whole is thin on its outer half, translucent at parts, shelving outwards to a sharp edge like an oyster shell; under surface marked by shallow depressions; upper surface irregularly grooved transversely, especially on outer half opposite the excavations (thickness at the grooves  $\frac{1}{12}$ , at the ridges  $\frac{3}{12}$  to  $\frac{2}{12}$ ). No ositic roughness on either surface. The peaks to which the ridges run outwards appear to represent, so far, the points to which the inter-muscular septa of the deltoid are attached. Length of entire acromion  $1\frac{9}{12}$ , breadth at most projecting peak  $1\frac{3}{12}$ . *Ossicle*, length  $\frac{9}{12}$ , breadth at base  $1\frac{1}{12}$ ; the breadth in this case, if not increased by disease, thus exceeding the length.

*Intra-acromial joint*,—on outer two-thirds, only a loose membranous ligament,  $\frac{2}{12}$  in length when stretched, connecting the thin edges of the bones; on inner third, the bones are in contact ( $\frac{1}{12} \times \frac{1}{12}$ ) with ligament above, below and internally, are lined by thin cartilage with appearance of a synovial cavity. The ossicle is very loose in all directions, and moves outwards and inwards for  $\frac{2}{12}$  inch when pushed or drawn by the clavicle.

*Acromio-clavicular joint*,—connexion only by a slack ligament ( $\frac{1}{3}$  to  $\frac{1}{2}$  inch in length,  $\frac{3}{4}$  in breadth,  $\frac{1}{12}$  in thickness) between the ends of the bones. Where ligament attached, ossicle  $\frac{1}{4}$  inch in thickness at middle, thin before and behind, concave both ways; end of clavicle  $\frac{3}{4}$  inch in breadth,  $\frac{1}{4}$  in thickness at middle, excavated both ways; no adaptation between the bones possible. The ligament has no attachment to the basi-acromion. Outward dragging of the ossicle by the action of the deltoid would be checked by the acromio-clavicular and coraco-clavicular ligaments, forward dragging by the intra-acromial ligament.

*Case 3.* On both sides. From a female subject æt. 82. No disease on either side. Figured in the Plate, left fig. 6, right fig. 6a.

*Scapulæ* slender, thin and very translucent on both fossæ, ridges on subscapular fossa pretty well marked. Both scapulæ, length  $5\frac{3}{4}$ , breadth  $3\frac{3}{4}$ . *Acromion*, form on the two sides, and that of the ossicle, seen in figs. 6 and 6a. The form is between the ovoid and crescentic types, the outer border much bent at and near the intra-acromial joint, the posterior angle somewhat rounded, the anterior end blunt-pointed. *Ossicles*, triangular with blunted apex; left longer than right, forming more than half of entire acromion, the right less than half. Length of both acromions  $1\frac{9}{12}$ ; breadths at middle of basi-acromion, right  $1\frac{1}{12}$ , left  $1\frac{9}{12}$ . Breadth of both ossicles at base  $\frac{9}{12}$ ; lengths, right  $\frac{9}{12}$ , left 1 inch. The left ossicle, the longer one, shows two deltoid tubercles, the basi-acromion one tubercle close to the joint; the right ossicle one elevation, the basi-acromion two. There are besides the tubercles represented by the anterior and posterior angles.

*Intra-acromial joint*, good ligament above and below, the former rather the stronger, attached to raised edges above and below on both bones. These ligaments unite round the borders, on inner border forming a septum between the intra-acromial and the acromio-clavicular synovial cavities. Movement, though limited by these ligaments, is enough to be recognisable in the living body. Synovial cavity present on both sides throughout; surfaces not smooth and polished like ordinary articular cartilage, but finely mottled; the covering of cartilage thin. Depth of the surfaces at the middle  $\frac{1}{12}$ , being greater than

the thickness of the ossicle and basi-acromion ( $\frac{3}{12}$ ), owing to the raised edges at the joint. In direction, the joints differ on the right and left sides. The way generally in the series of specimens is, that the basi-acromion forms a projection and falls away backwards on either side, giving a general concavity backwards, with, it may be, minor undulations. That is seen here on the right side (fig. 6a), the projection being external to the middle; but on the left side (fig. 6) that on which the ossicle is the longest, it is the reverse, the projection being on the ossicle, and the outer slope of the joint has a forward instead of the usual backward direction.

*Acromio-clavicular joint.*—It is worthy of note that the inferior ligament, normally very slender, is stronger than usual. In both scapulæ the clavicle rests entirely on the ossicle, as shown in the figures, the connexion of the clavicle to the basi-acromion being only by the posterior part of the acromio-clavicular ligaments. Length of acromio-clavicular surfaces,  $\frac{1}{2}$  inch, height  $\frac{3}{8}$  inch; length being thus the same on both sides although the left ossicle is longer than the right. In both the joint begins at the posterior part of the ossicle. Acromial facet concave both ways, clavicular facet convex both ways; joint has the usual slope downwards and inwards. The acromial facet is lined by smooth articular cartilage. In the upper half of the joint on the right side there is a *true incomplete inter-articular fibro-cartilage*, elliptical and wedge-like (height  $\frac{2}{12}$ , length  $\frac{1}{2}$  inch) attached above to the superior ligament, below with natural sharp edge. Its cartilage-like appearance marks it off distinctly from the fibrous tissue of the ligament from which it hangs into the joint. In the left joint it is much less apparent, about half the height of the one in the right joint, and with fringed free edge, hardly an advance on the synovial tufts that usually hang into the joint from above and are liable to be mistaken for a partial inter-articular fibro-cartilage.

Sockets of shoulder joint healthy, except some thickening of long tendon of biceps at its origin in left shoulder.

*Case 4.* From a female subject æt. 80. Left scapula; no disease. Right not noted.

*Scapula*,— $5\frac{1}{2} \times 3\frac{3}{4}$  inches; very thin and translucent at fossæ; subscapular ridges very faint, but other muscular markings well developed. *Acromion*, crescentic type, posterior angle rounded off, outer border much bent at and behind intra-acromial joint; point does not project beyond line of clavicle. Length of entire acromion  $1\frac{6}{12}$ , breadth of basi-acromion at middle  $1\frac{1}{2}$ . *Ossicle*, length  $1\frac{10}{12}$ , breadth at base  $1\frac{10}{12}$ . Outer border shows no deltoid tubercle, but projects considerably at the base; basi-acromion shows two tubercles, besides the posterior angle, one of them at the joint.

*Intra-acromial joint*, ligament above and below, attached to raised edges, joining at outer and inner sides, the inferior ligament quite as strong as the superior. Movement quite sufficient to be recognisable in living body. Synovial cavity throughout, and not continuous with acromio-clavicular joint; surfaces ( $1\frac{10}{12} \times \frac{5}{12}$ ) same appearance as noted above in other specimens, the covering of cartilage thin and with smooth but finely mottled surface. Direction of joint, at right angles to axis of acromion, undulating but nearly transverse generally, outer fourth inclined moderately backwards.

*Acromio-clavicular joint.*—Clavicle rests entirely on ossicle; connected to basi-acromion only by posterior part of ligaments. Inferior ligament, as in case 3, much stronger than normal. Facets, length  $\frac{6}{12}$ , height  $\frac{3}{12}$ , the acromial one concave both ways, the clavicular one convex both ways; have the usual slope downwards and inwards. Surface of acromial facet smooth and polished articular cartilage, that of clavicular facet smooth but tending to spongy condition. A *complete inter-articular fibro-cartilage* present, with completed synovial cavity on each side of it. It is attached all round, below to the clavicle rather than to the ligament; is about  $\frac{1}{24}$  inch in thickness, semi-transparent, but firm enough to maintain the curvatures that had adapted it to the

facets. This is the only instance of the occurrence of a complete inter-articular fibro-cartilage at this joint I have ever seen.

The socket of the shoulder joint is healthy ; also the tendon of the biceps.

*Case 5.* From a male subject æt. 64. Right scapula ; no disease. Some appearance of corresponding groove on left acromion.

Length of right *scapula*  $6\frac{1}{2}$  inches, of left  $6\frac{5}{8}$  ; breadth of both  $4\frac{1}{8}$  ; both have strong muscular markings. *Right acromion*, form rather of the oval type, posterior angle rounded, anterior end a blunt point projecting a little ( $\frac{1}{8}$  inch) beyond clavicle, outer border considerably convex at and near intra-acromial joint. Length 2 inches, breadth of basi-acromion 1 inch. *Ossicle*, length  $1\frac{1}{2}$ , breadth at base 1 inch. Outer border of ossicle shows a low elevation and projects considerably at the base ; basi-acromion shows two deltoid tubercles, one near the front and one in front of the rounded angle.

*Intra-acromial joint*.—Ligament above and below attached to raised lips ; the lips above considerably raised, suggesting slight ostitic action here. Amount of movement allowed should be quite recognisable in living body, especially depression. Synovial cavity throughout ; surfaces, as noted in previous cases, smooth and mottled and layer of covering cartilage thin. Surfaces, 1 inch by  $\frac{1}{2}$  inch ; upper crest and greater convexity above give unusual height to the surfaces. Thickness of basi-acromion and of ossicle  $\frac{1}{2}$ , thickness (height) at the raised lips  $\frac{1}{2}$ . Direction of the joint, nearly transverse, with slight general concavity of basi-acromion ; no backward direction of joint at outer side.

*Acromio-clavicular joint*.—Clavicle rests entirely on ossicle, posterior part of ligaments alone connecting clavicle to basi-acromion. Inferior ligament stronger than usual. Facets, length  $\frac{7}{12}$ , height  $\frac{1}{12}$  ; acromial concave both ways, clavicular convex both ways ; joint has usual slope downwards and inwards ; cartilage of both smooth, that of clavicle thicker, showing tendency to spongy condition. Synovial tufts hang into joint from above, simulating a partial fibro-cartilage, but are only ragged tufts.

*Left acromion*.—On upper surface a shallow groove crosses the acromion in nearly same position as the intra-acromial joint of right acromion, almost exactly in same position except that it bends back at the outer and inner sides. Is best marked on inner half, its sharp posterior boundary prolonged from upper edge of spine of scapula. As the groove is not deep and as there is no groove or mark on the under surface, I hesitate to consider it a vestige of the union of a formerly separate ossicle, but it may be so. Acromio-clavicular joint of left side, same as noted on right side. Form of acromion, like the right, but wants the considerable convexity of outer border seen on the right acromion at and near the intra-acromial joint. Of same breadth as the right, it is  $\frac{2}{12}$  shorter, which might accord with the disappearance of a former intra-acromial joint. Of three tubercles seen on outer border of acromion between anterior end and posterior angle, two are in front of the groove, one of these close to it ; the third is behind the groove on what would have been the basi-acromion.

#### (B.) MACERATED SPECIMENS.

The following are macerated specimens in my collection, with the exception of Nos. 12 and 13, which are in the Charles Bell collection in the Museum of the College of Surgeons. They are all adult. The sex can be only inferred from the size and muscularity, and consequently appears in the Table (Table II.) as either entirely uncertain or as probable. Of these eight specimens six

are of the left side ; there is no record of the condition of the other side.

*Case 6.* Fig. 7. Left scapula ; no appearance of disease.

*Scapula*, length 6 inches, breadth  $3\frac{2}{3}$ , muscularity moderate. *Acromion* mostly of the ovoid type, posterior angle rounded, length of entire acromion  $1\frac{1}{2}$ , breadth at middle of basi-acromion 1 inch. *Ossicle*, length  $\frac{1}{2}$ , breadth at base  $\frac{1}{2}$ . Two of the tendon tubercles seen on the ossicle, the second of these at its base ; basi-acromion scarcely shows a tubercle. *Intra-acromial joint*, line wavy, general direction nearly transverse to axis of acromion, but outer third directed obliquely backwards. *Acromio-clavicular joint*, facet for clavicle, length  $\frac{9}{12}$ , of which about a fourth part is on basi-acromion. The figure (fig. 7) is given as an example of the partial resting of the clavicle on the basi-acromion, although that is not owing to shortness of the ossicle.

*Case 7.* Fig. 8. Left scapula ; no appearance of disease. From size and muscularity inferred to be probably male.

*Scapula*, length  $6\frac{3}{8}$  inches, breadth 4 ; muscular. *Acromion*, of the triangular type, antero-external angle wanting, antero-internal angle very pointed and prolonged a little ( $\frac{1}{8}$  inch) beyond clavicle ; length of entire acromion 2 inches, breadth of basi-acromion  $1\frac{2}{12}$ . *Ossicle*, long ; length  $1\frac{3}{12}$ , being longer than basi-acromion by  $\frac{1}{2}$  inch ; breadth at base 1 inch. Tendon tubercles on outer border of ossicle rather indefinite, but appearance of two, and base projects ; one tubercle, perhaps two, on basi-acromion, besides its angle. *Intra-acromial joint*, line sigmoid, on basi-acromion concave internally, convex externally, then directed a little backwards ; general direction a little outwards and backwards. Lips of joint project a good deal above. *Acromio-clavicular joint*, facet entirely on ossicle, length fully  $\frac{9}{12}$  inch ; is about  $\frac{1}{2}$  inch from base and from apex of ossicle.

*Case 8.* Left scapula ; no appearance of disease.

*Scapula*, length  $5\frac{6}{8}$  inches, breadth 4 ; apparently aged, fossæ very translucent and muscular ridges little marked. *Acromion*, like No. 7, of the triangular type, considerable convexity on outer border at the joint ; length of entire acromion  $1\frac{6}{12}$ , breadth of basi-acromion 1 inch. *Ossicle*, length  $\frac{9}{12}$ , being equal to that of basi-acromion, breadth at base 1 inch. The considerable convexity on outer border at and near joint appears as if formed by a tendon tubercle on each bone ; anterior tubercle on ossicle indefinite ; posterior angle of basi-acromion fairly well marked. *Intra-acromial joint*, general direction outwards and a little backwards ; inner two-thirds wavy, obliquely backwards at inner side ; outer third obliquely backwards till at margin. *Acromio-clavicular joint*, facet entirely on ossicle ; length  $\frac{6}{12}$ , height  $\frac{3}{12}$  ; extends from near base of ossicle to  $\frac{3}{12}$  inch from point ; surface foraminated. Both basi-acromion and ossicle thin in this apparently aged specimen.

*Case 9.* Left scapula ; no appearance of disease.

*Scapula*, part below spine and glenoid cavity has been sawn off ; breadth  $3\frac{1}{2}$  inches ; muscular markings on spine moderate. *Acromion*, mostly of the ovoid type, posterior angle well defined behind but not very prominent, outer border considerably bent at the joint. Length of entire acromion  $1\frac{8}{12}$ , breadth of basi-acromion 1 inch. *Ossicle*, length 1 inch, being longer than basi-acromion by  $\frac{1}{3}$  inch ; breadth at base 1 inch. On its very convex outer border two low tendon tubercles seen and base projects. Slight tendon tubercle seen at middle of short basi-acromion. *Intra-acromial joint*, angular, inner third directed backwards, outer two-thirds backwards till just at margin, general

direction backwards and outwards. *Acromio-clavicular joint*, facet entirely on ossicle, might seem partly on basi-acromion but not so, triangular area behind it is foraminated and facet seen to be finished at back part of ossicle; length of facet  $\frac{7}{12}$ , height at middle  $\frac{4}{12}$ ; at front of facet ossicle slopes off to blunt apex; direction of facet mostly downwards and outwards (not inwards).<sup>1</sup>

*Case 10.* Left scapula; no appearance of disease.

*Scapula*, length  $6\frac{3}{8}$  inches, breadth  $3\frac{2}{8}$ , narrow for its length; not muscular. *Acromion*, of the crescentic type, posterior angle quite rounded off obliquely, only a very low projection to mark off acromion proper from expanding crest of spine; outer margin uniformly convex, antero-external as well as posterior angle wanting, giving the crescentic form; anterior end pointed but not sharp; length of entire acromion,  $1\frac{8}{12}$ , breadth of basi-acromion 1 inch. *Ossicle*, length  $\frac{8}{12}$ , breadth at base  $\frac{9}{12}$ . Two low tendon tubercles on ossicle and projection at joint; on basi-acromion one tubercle besides projection at joint. *Intra-acromial joint*, angular, outer third directed moderately backwards, inner two-thirds directed more obliquely backwards; upper edges considerably raised. *Acromio-clavicular joint*, facet rests partly on basi-acromion, height  $\frac{4}{12}$ , length  $\frac{7}{12}$ , of which  $\frac{1}{2}$  is on basi-acromion; anterior end is  $\frac{3}{12}$  from point of ossicle.

*Case 11.* Right scapula; two ossicles, anterior and posterior, leaving the acromion in three pieces; traces of chronic arthritis on under surface of the ossicles. Fig. 9.

*Scapula*, only outer portion with the processes and outer part of clavicle present; evidently a strong muscular scapula. *Acromion*, of the crescentic type, posterior angle moderately rounded, outer margin uniformly convex, pointed in front, giving crescentic form. Length of entire acromion  $2\frac{9}{12}$  inches, of which  $1\frac{5}{12}$  formed by basi-acromion; breadth of basi-acromion  $1\frac{5}{12}$ . *Two epi-acromial ossicles*; *posterior*, length 1 inch, breadth at base  $1\frac{1}{12}$ ; *anterior*, length  $\frac{5}{12}$ , breadth at base  $\frac{6}{12}$ , triangular and pointed at end. *Posterior intra-acromial joint*, general direction outwards and forwards, wavy, slopes backwards more at inner than at outer side. *Anterior intra-acromial joint*, general direction nearly transverse. Tubercles on outer border of acromion indefinite, two on basi-acromion besides posterior angle, the front one at the middle, and projection at joint; one on posterior ossicle besides projection at base, but border serrated; tubercles on anterior ossicle represented by point and by projection at base. *Acromio-clavicular joint*, clavicle rests entirely on posterior epi-acromial ossicle; posterior part of ligaments attached to basi-acromion, anterior part attached to base of anterior ossicle.

*Amount of diseased appearance.*—On upper surface, edges of both intra-acromial joints raised more than usual and surface of posterior ossicle rather rough; on under aspect, posterior ossicle has smooth polished "porcellanous" surface finely foraminated, anterior ossicle a little rough but not porcellanous.

<sup>1</sup> *Note on ossification of the supra-scapular ligament.*—In evidence that there was no want of tendency to ossification in this scapula, may be noted that the supra-scapular ligament is represented by a strong bridge of bone,  $\frac{1}{2}$  inch in breadth. In three other scapulæ in my collection having this bridge of bone (all from different subjects), one, a left, has the bridge as broad as in case 9; in the other two, right scapulæ, the bridge is only about  $\frac{1}{4}$  inch in breadth. In one of the latter the foramen is elongated and slightly hour-glass shaped, and would take in both the nerve and the bloodvessels; in the two with the broad ridge not more than the nerve could pass through. These bridges of bone, usually described as resulting from ossification of the ligament, are quite smooth bone.



No other appearance of disease ; glenoid cavity healthy and tendon of biceps normal.

The subdivision of the epi-acromion in this case might have been attributed to disease had there been marked arthritis, but as the shoulder joint is healthy and as the appearance of disease seen on the posterior ossicle is not great, the conclusion appears to be that we have here a case in which the ossific centres for the anterior part of the acromion have remained separate as well as a case in which the major epi-acromion has remained separate from the basi-acromion.

I am indebted to the Museum Committee of the College of Surgeons for permission to notice the two following specimens, Nos. 12 and 13.

*Case 12.* Left scapula showing separate epi-acromion, and appearance as if of united fracture at lower angle and base of bone. No appearance of disease.

The specimen, No. 3, 102 in the printed catalogue, is marked "Ununited fracture of the acromion process and united fracture of the vertebral border." In the MS. catalogue of Sir Charles Bell the description is, "There has been a fracture of the acromion scapulæ ; a false joint must have been formed between the two portions ; at the lower angle a fracture, or perhaps a diastasis of the cartilage, has taken place, which is now united." I mention this to show the view that used to be taken of the nature of this condition of the acromion. The acromion, however, presents the usual family likeness of cases of separate epi-acromion.

*Scapula*, moderately muscular and of good size, length  $5\frac{1}{4}$  inches, breadth 4 (somewhat diminished by the supposed fracture). The line of the seeming fracture cuts off  $\frac{1}{2}$  to  $\frac{3}{4}$  inch at lower angle and about  $\frac{1}{2}$  inch of the breadth of the base up to nearly midway to the spine. The united part is inclined to the subscapular fossa, and the fracture thus appears to have been caused by a force from behind. No appearance of other injury to the bone and none of disease. *Acromion*, form indefinite, between the crescentic and triangular types, posterior angle much sloped off, almost continued from lower edge of crest of spine, anterior end blunt-pointed, general triangular form given mainly by form and great size of epi-acromion. Length of entire acromion  $1\frac{6}{12}$ , of which only the  $\frac{1}{12}$  formed by basi-acromion ; breadth  $\frac{9}{12}$ . *Ossicle*, length 1 inch, breadth at base (the broadest part of entire acromion) 1 inch. Tubercles on outer border of acromion indefinite, one elevation on epi-acromion, a slight one on basi-acromion, and both have projecting angles where they meet at the joint.

*Intra-acromial joint*, general direction transverse, but with concavity backwards ; margins a little raised except on ossicle below. *Acromio-clavicular joint*, entirely on ossicle, facet on ossicle ovoid and short, length  $\frac{6}{12}$ , height  $\frac{3}{12}$ , situated  $\frac{3}{12}$  from posterior end of inner border of ossicle,  $\frac{4}{12}$  from anterior end ; has very little inclination downwards and inwards.

*Case 13.*—Right scapula in which a formerly separate ossicle is now mostly united to the basi-acromion. This specimen is exceptionally interesting as showing union taking place. No appearance of disease.

In the original Bell catalogue the specimen is marked as "A fracture of the acromion scapulæ." In the valuable recent printed catalogue, 1893, by the

Conservator of the Museum, Mr Cathcart, it is marked "No. 3, 103. Supposed fracture of the acromion process of the scapula. Right scapula—macerated—showing an irregular groove between the acromion process and the rest of the bone. This is possibly a late union of the epiphysis."

*Scapula* large and muscular, in all probability male; length  $6\frac{1}{2}$  inches, breadth 4. *Acromion*, of bluntly crescentic form, inner border very little concavity, external border uniformly convex, anterior end blunt, posterior angle very little marked. Length of entire acromion 2 inches, breadth  $1\frac{1}{2}$ . *Ossicle*, length  $1\frac{1}{2}$ , breadth at base 1 inch. Tubercles on outer edge of acromion, on the ossicle two besides point and corner at suture; on basi-acromion, one very low tubercle at middle, and corner at suture.

*Intra-acromial suture*, direction nearly transverse, slightly outwards and backwards; nearly straight. The suture of union is strongly marked above, as a furrow about  $\frac{1}{2}$  inch wide and nearly the same in depth; its anterior boundary sharp and overhanging, the posterior boundary bevelled with slightly raised margin; floor of furrow shows a few fine foramina. On under surface only a shallow furrow seen, with corresponding direction.

*Acromio-clavicular joint*.—A very small part of the facet, hardly  $\frac{1}{12}$  inch, is on the basi-acromion; length of facet  $\frac{5}{12}$ , height  $\frac{1}{12}$ , distance from anterior end of inner border of ossicle  $\frac{3}{12}$ .

The probable interpretation in this case is that the union of the epiphysis has been delayed. If it has been a case of fracture, the fracture has taken place at the usual line of epiphysial union, and is undergoing bony union without callus.

#### IV. CONSIDERATION OF THE CAUSES OF THE CONDITION OF SEPARATE ACROMION PROCESS.

##### *Fracture. Relation to Chronic Rheumatoid Arthritis. Relation to the Epiphysis.*

It is not easy to find one's way to definite conclusions on these relations amid the numerous and conflicting opinions to be found in the literature of the subject.<sup>1</sup>

<sup>1</sup> General statements in the Text-books of Surgery need not be referred to when not bearing evidence of being founded on personal observation. The following are the writings of authors more particularly referred to:—The late *Prof. Robert Adams*, of Dublin—(a) "Shoulder Joint, Abnormal Conditions of," in *The Cyclopaedia of Anatomy and Physiology*, vol. iv., article written in 1849; and (b) *Treatise on Rheumatic Gout*, 2nd ed., 1873, with accompanying vol. of Plates. The late *Prof. Robert W. Smith*, of Dublin, "Observations upon Chronic Rheumatic Arthritis of the Shoulder," in the *Dublin Quarterly Journal of Medical Science*, February and May 1853. *Mr John Gregory Smith*, "Pathological Appearances in Seven Cases of Injury of the Shoulder Joint, with Remarks," in *The London Medical Gazette*, vol. xiv., 1834. *Mr W. Arbuthnot Lane*—(a) "Some Points in the Physiology and Pathology of the Changes produced by Pressure in the Bony Skeleton of the Trunk and Shoulder Girdle," in *Guy's Hospital Reports*, vol. xliii., 1886; (b) "The Causation and Pathology of the so-called disease Rheumatoid Arthritis, and of Senile Changes," in *Transactions of the Pathological Society of London*, vol. xxxvii., 1886; and (c) "Mode of Fixation of the Scapula, etc.," in the *British Medical Journal*, 19th May 1888. *Prof. F. H. Hamilton*, of New York, *Practical Treatise on Fractures and Dislocations*, 8th ed., revised and edited by *Prof. Stephen Smith*, of New York, 1891.

## (A.) FRACTURES OF THE ACROMION.

*Question of frequency. Distinction between true fracture and conditions simulating fracture.*

The impression that fracture of the acromion process is not a very common occurrence appears to be conveyed by *Sir Astley Cooper's* opening words on this fracture in his celebrated *Treatise on Dislocations and Fractures of the Joints*—"This point of bone is sometimes broken." But from the remarks that follow, in regard to the detection of the fracture, it is evident that this great surgeon and anatomist was familiar with the occurrence. He speaks of diminished roundness of the shoulder, of feeling the depression on tracing from the spine to the clavicle, and of crepitus being felt when the surgeon places his hand upon the acromion and rotates the arm. In the accompanying dorsal diagrammatic figure (p. 411, new ed., 1842) the line of fracture has the family likeness to the specimens I have described of separate acromion. Of the second figure given (p. 412), he says:—

"This figure shows a fractured acromion; the edges of the fractured surfaces are united by ligament, part of which has been turned aside to show ligamentous granulations upon the fractured surface." No history is given for the preparation. The figure is a front view, and it also has the family likeness to my specimens. He adds—"Fracture of the acromion may unite by bone; but it generally unites by ligament, in consequence of the difficulty which exists in producing adaptation and in preserving the limb perfectly at rest during the period required for union." He does not allude to fracture without displacement, and consequently assumes difficulty in producing adaptation.

It is not evident but that his remark just quoted, as to non-union being general, may be founded on dissecting-room specimens of separate acromion. He makes no reference to the epiphysis.

*Sir William Fergusson*, writing some years later (*Practical Surgery*, p. 185, 1842), says:—

"The acromion process may be broken, but the accident is of rare occurrence. I have dissected a number of examples of apparent fracture of the end of this process; but in such instances it is doubtful if the movable portion had ever been fixed to the rest of the bone." In the figure he gives to show "the ordinary position of some of the fractures here referred to," the line on the acromion gives more to the broken off part than in my specimens, but the figure is diagrammatic.

Thus Fergusson, thorough surgical anatomist as well as experienced surgeon, was in doubt whether such specimens should be regarded as cases of fracture or as cases of non-union of the epiphysis.

*Prof. Adams*, under the head of "Fracture of the acromion process" (*loc. cit.*, a, p. 600), remarks:—

"The fracture of the acromion will be generally found to have taken place at a point behind, and within, the junction of the clavicle with this bony process; its direction we always observe to be in the original line of the junction of the epiphysis with the rest of the bone."

No cases are given, and the above remark is too general. He is probably influenced by his favourite view, alluded to below, of separation taking place at the line of junction of the epiphysis in cases of chronic rheumatic arthritis; and in his subsequent references to the opinions of Sir A. Cooper and Malgaigne the reader is left in doubt whether the remarks of these authors are founded on museum specimens, such as those described in this paper, or on cases ascertained to have been traumatic fractures.

The view expressed by *Prof. F. H. Hamilton (loc. cit., p. 199)*, a recognised authority on fractures and dislocations, may be taken as his judgment on the evidence submitted by previous writers as well as embodying his own experience, and they have largely influenced modern writing and teaching on this question.

“There is some reason to believe, I think, that a true fracture of the acromion process is much more rare than surgeons have supposed, and that in a considerable number of the cases reported there was merely a separation of the epiphysis; the bony union having never been completed. If such fractures or separations occurred only in children, very little doubt might remain as to the general character of the accident; but the specimens which I have found in the museums, and the cases reported in the books, have been mostly from adults. It is more difficult, therefore, to suppose these to be examples of separation of epiphyses, but I am inclined to think that in a majority of instances such has been the fact. It is very probable, also, that in the case of many of the specimens found in the museums, called fractures, the histories of which are unknown, they were united originally by cartilage, and that in the process of boiling, or of maceration, the disjunction has been completed. The narrow crest of elevated bone which frequently surrounds the process at the point of separation, and which Malgaigne may have mistaken for callus, is found upon very many examples of undoubted epiphyseal separations which I have examined; and this circumstance, no doubt, has tended to strengthen the suspicion that these were cases of fracture.”

“There is no doubt, however, that a fracture of this process does occasionally take place. Examples of fracture of the acromion process have been reported by Duverney, Bichat, Avrard, A. Cooper, Desault, Sanson, Nélaton, Malgaigne, West, Brainard, Stephen Smith, and others. *I have myself seen five cases.* In the case seen by Cooper it entered the articulation of the clavicle, and produced at the same moment a dislocation. Malgaigne says it occurs generally further up, and *posterior to the attachments of the clavicle*, ‘near the junction of the diaphysis with the epiphysis,’ and that the fracture is in most cases transverse and vertical; but Nélaton saw a case in which the fracture was oblique. In the case reported by C. West, of Hagarstown, Md., the fracture was *through the base of the process.* In two of the examples seen by me the fracture was *in front of the clavicle*; in the *third*, occasioned by the fall of a barrel of flour upon the shoulder, the fracture occurred at the *acromio-clavicular articulation*, and was accompanied with an upward dislocation of the outer end of the clavicle; in the *fourth*, the fracture occurred at the same point, but there was *neither displacement of the clavicle nor of the process*, the fracture being only recognised by crepitus and motion. The *fifth* was brought to my notice by Dr Sabine, surgeon to Bellevue Hospital. The patient had been struck by a policeman’s club. There was distinct crepitus, the fracture being *posterior to the acromio-clavicular junction*, but there was *no displacement* of the fragments or of the clavicle. Some of the fractures were confirmed by dissection, and in the case mentioned by Dr Stephen Smith, an autopsy, made three weeks after the accident, showed a fracture *in front of the clavicle without displacement*, the periosteum covering its upper surface not being torn; the fragment could be

turned back as upon a hinge. [This fracture was caused by a blast; a fragment of rock being driven upward struck the acromion on the under surface.]”

I have placed some of the words in the above quotation in italics in order to call attention to the localities of the fracture. In the five cases of undoubted traumatic fracture that came under Hamilton's personal observation, two of them (and Dr Stephen Smith's case) were in front of the clavicular connexion; two of them at the acromio-clavicular articulation (meaning, we may infer, opposite some part of the articulation); in the fifth case the fracture was behind the clavicular connexion. There was no displacement in one of the cases (the fourth) in which the fracture occurred opposite the clavicular connexion, nor in the one (the fifth) behind that connexion, nor in Dr Stephen Smith's case where it occurred in front of the clavicle. From this undoubted, though limited, statistic we see that traumatic fracture may occur at any part of the acromion, depending, no doubt, on the kind and locality of the force; further, that there may be no displacement, but that, notwithstanding, the existence of the fracture may be detected by careful examination.

These references sufficiently show that in the opinion of Hamilton traumatic fracture is a comparatively rare occurrence, so that, although alive to the question and expert in the mode of its detection, he could, in his wide experience, refer to only five cases of the occurrence.

In strong contrast with the usually accepted opinion of Hamilton, is that of *Mr Arbuthnot Lane*, of Guy's Hospital, who holds that fracture of the acromion process is the most common of all fractures. In 325 bodies whose bones he carefully examined in the dissecting-room (*loc. cit.*, c, 1888, p. 1048), while he found no instance of undoubted fracture of the coracoid process, he “found that a considerable proportion presented fractures of the acromion, that portion of the scapula being broken more frequently than any other bone in the body.” He refers to the statistics of fractures of the upper extremity treated at the Middlesex Hospital in ten years ending 1879, a total of 1084 fractures, of which 6 were of the coracoid process and only 10 of the acromion. This contrast to his own finding in the dissecting-room he attributes to “the comparative difficulty in detecting a fracture of the acromion. I have in many instances of so-called simple contusion of the shoulder of the living subject been able to satisfy myself of the presence of an ununited fracture of the acromion or of fracture of the outer third of the clavicle.”

In a previous paper (*loc. cit.*, a, 1886) Mr Lane includes a full consideration of the question of the frequency of fractures of the acromion. The paper is an elaborate and thoughtful one, embracing the consideration of the trunk and shoulder girdle, and contains much original matter with pretty strong criticism of the views of

other writers. Mr Lane's paper is a valuable contribution to the discussion of the interpretation of the condition of separate acromion process, but requires to be read critically. He assumes too readily that all such specimens, whether with or without the accompaniment of rheumatoid arthritis, were originally cases of fracture, and he appears not to be aware of the frequency of the occurrence of the separation at the locality of the union of the epiphysis.

Experimenting on the dead body, Mr Lane finds (p. 408):—

“If the outer end of the shoulder as represented by the acromion be struck vertically, obliquely, or at right angles to the vertical by a light hammer the acromion is fractured with the greatest facility. This fracture usually corresponds in position with the seat of fracture as found pathologically, and its direction will be found to vary within the same limits. In this artificial fracture the line of fracture may pass in front of or behind the oval clavicular facet or through any portion of its area. Using the same light hammer, but with greater force, the clavicle may be broken. It is broken much more easily in that portion of its extent which lies outside the conoid tubercle.”

Referring to his observation of the bones in numerous subjects in the dissecting-room, Mr Lane says (p. 415):—

“I found a great many instances of fractures of the acromion, but did not consider this unnatural, judging from the facility with which I was able to break the acromion with a blow applied horizontally as in a fall on the shoulder, or vertically as when the shoulder is struck by a falling body. In many of those in which the fracture was in front of the clavicular facet I found considerable displacement of the fragments. The direction and seat of the fracture varied within a broad limit, corresponding to the direction of the fractures I obtained experimentally on the dead body. In some cases the fragments were connected by intervening fibrous tissue, while in other cases they were united by a fibrous capsule lined by a synovial membrane. The latter arthrodial joint allowed of much more freedom of movement than the amphiarthrodial joint, and the extent of development present in the newly-formed articulation was evidently dependent on the amount of movement to which it had been subjected by the occupation of the individual and to a less degree upon the direction and seat of the fracture.”

“In almost all these cases *the shoulder joints presented the changes which are usually described as rheumatoid arthritis*, but which I think are better described as *pressure changes*. These, I am convinced, must have been consequent on the injury that caused the fracture of the acromion, so that the fracture and the joint changes were both expressions of the blow received, which was the cause of both.”

It will have been observed by the critical reader that in these quotations there is a good deal of theory and general statement, and ready assumption that all of the separate acromions found in the dissecting-room had been cases of fracture. It need not be doubted that the acromion may be readily broken in the dead body, or that it may, and probably is, often broken in the living body, at any place; but the question is, Why the separation is so often found just behind the clavicular connexion, at the place of union of the epiphysis, and how that is to be explained; and as bearing on the supposed connexion with rheumatoid arthritis it is necessary to be informed in each case to what extent the head of the humerus had eburnated or excavated the acromion.

Mr Lane notes (p. 416) five museum specimens, all of which he assumes to have been traumatic fractures, and which he regards as "fair instances of ununited fracture of the acromion."

In Nos. 1, 3, and 4 there are "extensive so-called rheumatoid changes in the shoulder joint," but to what extent the under surface of the acromion was eburnated or excavated is not stated. The lines of separation are various in position and direction. They proceed outwards:—In No. 2, "from a point behind the clavicular facet. The surfaces of the fragments are smooth and eburnated, and are connected by a capsule lined by synovial membrane. The glenoid cavity presents but slight change." In No. 4, "from a point just behind the clavicular facet. The union is by dense ligamentous tissues. There is no displacement of fragments." In No. 5, "outwards and backwards from a point just in front of the clavicular facet. The fragment is displaced somewhat downwards and forwards. The surfaces are united by dense ligamentous tissue." In No. 1, "outwards from just behind the anterior margin of the clavicular facet. The outer fragment is connected to the acromion by ligamentous tissue; there is no synovial cavity. It is displaced forwards in a horizontal plane." In No. 3, "outwards from the centre of the clavicular facet. There is no displacement, and the fragments are united by dense ligamentous tissue."

It will be observed that in the two cases first quoted (Nos. 2 and 4) the *line of separation* was just behind the clavicular facet, the position at or close to which it occurred in all of the thirteen cases I have described, being the locality of the union of the epiphysis. The other three cases, from the localities of the separation, could only be cases of true fracture.

In explanation of the *displacement* noted in Nos. 1 and 5, Mr Lane remarks (p. 421):—

"The forward displacement of the fragment when the fracture occurs outside or through the anterior portion of the clavicular facet is due to the gradual contraction of the coraco-clavicular ligament which draws it downwards and forwards."

That may be, but the unresisted dragging down of that portion of the deltoid muscle would seem a better explanation. That there may be no displacement when the fracture is in front of the clavicle is seen in Dr Stephen Smith's case, above referred to. The periosteum was not torn, as probably enough it is not in most cases of fractured acromion; accounting for their being so often overlooked. There is no displacement in any of the thirteen specimens I have described; open to the alternative interpretation of epiphyseal separation or of fracture. On the latter supposition, if the action of the deltoid muscle and the contraction of the coraco-acromial ligament should cause displacement of a fractured anterior part of the acromion, would we not much more expect displacement in fracture at the post-clavicular line? The whole acromio-clavicular connexion is then detached from its basi-acromial support, the scapula now hung to the clavicle only by the coraco-clavicular ligaments, as in fractures near the outer end of the clavicle, in which considerable displacement is often enough seen.

Mr Lane notes two specimens of *united* fracture of the acromion (p. 417), both presenting evidence of severe injury to the shoulder.

In the first, there is also "an unreduced sub-coracoid dislocation of the head of the humerus;" in the second, the head of the humerus "is much deformed and shortened." In the first, "Its direction is outwards from the *centre of the clavicular facet*. The fracture has united firmly by bone. There is considerable horizontal displacement of the outer fragment, the angular intervals between it and the inner fragment being filled in by osseous material." In the second, "The direction of the fracture is outwards from a point a *quarter of an inch behind the clavicular facet*. The outer fragment slopes downwards and outwards. There is more callus uniting the fragments on the lower than on the upper surface of the fracture."

These appear to have been cases of true fracture, and followed by bony union. There was some displacement in both. In explanation of the occurrence of bony union in these two cases, Mr Lane says—"The complete rest of the parts necessitated by the more severe injury to the shoulder-joint in both these cases has permitted of the fragments of the acromion being kept sufficiently at rest to allow of their uniting by bone."<sup>1</sup>

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<sup>1</sup> To these I may add a case resembling the first of these two which, since writing the above, I have come upon in examining a preparation of old-standing unreduced sub-coracoid dislocation of the shoulder in the Charles Bell collection in the Museum of the Edinburgh College of Surgeons. The case has the further interest in that both shoulders had been dislocated. In the *left* shoulder (Cat. of 1893, p. 192, No. 5, 3), a spirit preparation that must have been put up more than sixty years ago, I find there has been fracture of the acromion with bony union; in the *right* shoulder, a dried preparation (No. 5, 4), the acromion is entire. As there is no history during life, it may be inferred that they were met with in the dissecting-room.

In the original MS. Bell Catalogue, the *left side* is described thus:—"Both arms of this man had been dislocated; on this side, the left side, the scapula has also been fractured through its body. The posterior portion has been dragged forwards by the action of the serratus magnus muscle, and the two portions have united irregularly; the newly formed joint of the shoulder is exposed." The *right side* is thus described:—"The right scapula and humerus from the same body as the preceding preparation, articulated as they were found in the body. There was dislocation inwards with fracture of the inner edge of the glenoid cavity. Imperfect ankylosis has taken place between the posterior part of the body of the humerus and that portion of the glenoid cavity which was fractured and was again united. The surfaces of the head of the humerus and of the glenoid cavity are both rough and unnatural, having for a long period before the patient's death been deprived of their office as a joint."

Both specimens show abundant evidence of rheumatoid action following the dislocations. They are true sub-coracoid dislocations, each with definite new glenoid cavity. The *right acromion*, a good-sized quadrate acromion, stands out prominently and is uninjured and healthy. The *left acromion* has been fractured just behind the clavicular connexion, the line directed outwards and a little forwards. There is no displacement. That there had been fracture I infer from the presence of a furrow on the upper surface and outer edge, about  $\frac{1}{8}$  inch in breadth and  $\frac{1}{2}$  to  $\frac{1}{10}$  inch in depth; and on the under surface from the presence of a callus-like ridge externally and internally with a furrow at the middle between them. These ridges led me to look for further evidence of fracture. The furrow above was seen only after removing the periosteum. The under surface of the acromion also was covered with periosteum. There is firm bony union.



In regard to the supposed rarity of the occurrence of fracture of the acromion, Mr Lane refers to the fact, above noted, that in the statistics of fracture treated at the Middlesex Hospital for ten years ending 1879, there were recognised only ten cases of fracture of the acromion; of which one occurred during the first five years of life, one between 15 and 30, four between 30 and 45, and the remaining four in patients above 45 years of age. And he remarks (p. 420):—

“These facts, taken in conjunction with the frequency with which this fracture is observed in the dissecting-room, and the facility with which it is artificially produced in the dead subject, serve to show *that in the living subject fractures of the acromion process are hardly ever diagnosed.*”

In support of his opinion that specimens of separate acromion have been cases of fracture, as opposed to the non-union of the epiphysis view, Mr Lane gives the following six reasons (p. 418):—

Against the possibility, as he expresses it, “that the bony centre in the extremity of the epiphysis has formed with the remainder of the bone an arthrodial or amphiarthrodial joint, as in the manubrio-sternal articulation and in the joint which usually exists between the body and cornua of the hyoid bone, I would put forward the following arguments :

1. The frequency with which it occurs associated with other fractures of the shoulder-girdle and its connexions, where it is extremely probable, if not absolutely certain, that it owed its presence to the same cause. In some of these cases an amphiarthrodial joint, with or without displacement, is present, while in others an arthrodial joint, with or without forward, or forward and downward, displacement exists.

2. The frequency of its association with so-called rheumatoid arthritis of the shoulder-joint, which I consider to be as much an expression of injury as fracture.

3. Its much greater frequency on the right side.

4. The broad limit in the space in which this condition is found. It may occur outside the clavicular facet or a considerable way inside.

5. The variation in the direction of the fracture with respect to the axis of the acromion.

6. The fact that the seats of this condition and those of fracture artificially produced differ in no single particular.”

In view of the facts and arguments submitted by Mr Lane, it is not possible to resist the conclusion that fracture of the acromion process is a much more frequent occurrence than had been supposed, or than is usually stated in text-books on surgery, and that the erroneous impression is owing to the difficulty in detecting the fracture, or, rather, to the readiness with which it may be overlooked. He is, however, not on so sure ground in holding that the “hypothesis of separation of the epiphysis” is “utterly without foundation.” He fails to account for the fact that, while fractures of the acromion may occur at any part, the usual place of separation is at or close to the post-clavicular line, the place of union of the epiphysis. That may be from his not having seen so many cases of its occurrence at that locality as I and others

have seen. That is the locality in all of the thirteen cases I have described, and I have not seen a specimen of its occurrence at any other part of the acromion. While not doubting that true fractures of the acromion occur at and in front of the clavicular connexion and that they may remain in the ununited condition, the fact that the usual locality of separate acromion is at the post-clavicular region remains for some anatomical explanation.

One of Mr Lane's list of reasons above quoted, the third, that has not been met in the preceding considerations, remains to be noticed:—"Its much greater frequency on the right side." That was so among the six cases noted by him; all of the five specimens given as of ununited fracture of the acromion were of the right side, while only the case of united fracture, accompanied by dislocation of the shoulder, was of the left side. The argument seems to have weight, taken along with the consideration that accidents to the upper limb, including dislocations of the shoulder, are more common on the right than on the left side. But turning to the facts given in my Table II., page 10, it is seen that of my 13 specimens, 8 were of the left side, 3 of the right side, 1 of both sides, and 1 (Nos. 5 and 6) certainly on the right side, doubtfully on the left. To these falls to be added the Charles Bell case (given in the footnote above) in which, with dislocation of the shoulder on both sides, the acromion had been fractured on the left side but not on the right. These numbers reverse Mr Lane's conclusion, and show the danger of founding a general conclusion on a limited statistic.

(B.) QUESTION OF THE CAUSE OF THE OCCURRENCE OF SEPARATE ACROMION PROCESS IN CASES OF RHEUMATOID DISEASE OF THE SHOULDER JOINT.

This co-existence was prominently brought into notice by the late Prof. Robert Adams of Dublin (*loc. cit.*), to whom, and to the late Prof. Robert W. Smith of Dublin (*loc. cit.*), surgery was indebted for original and valuable observations on the condition termed by Adams "chronic rheumatic arthritis," now usually called chronic rheumatoid arthritis.

The literature of the subject of chronic rheumatoid arthritis is extensive, and has involved the discussion of the occurrence of so-called "dislocation upwards" of the humerus, and the question is complicated by the frequent association of the rheumatoid condition with the appearances of old traumatic dislocation of the humerus forwards or downwards. Mr Adams labours to establish that the displacement upwards is not to be taken as primarily traumatic but as a secondary result of the rheumatoid disease, and that such secondary displacement may also take place inwards or downwards. Confusion has arisen from the term "new glenoid cavity" being vaguely or variously used. But it is

easy to determine that a specimen is one of old traumatic dislocation by the presence of a defined new glenoid cavity placed to the inside of or below the old cavity, sub-coracoid or sub-glenoid, where the dislocated head of the humerus had worked for itself a new cavity before the rheumatoid changes were set up. The new glenoid cavity so often mentioned by Adams is the space acquired upwards, by the gradual destruction of the capsular tendons and other soft parts, at length formed by the coraco-acromial arch and the old glenoid cavity together.

It is hardly necessary to say that traumatic dislocation of the humerus upwards is an anatomical impossibility unless the acromion process has been fractured and displaced upwards. The upper end of the humerus, cushioned by the capsular muscles, fully occupies not merely the glenoid cavity but also what may be called the accessory socket of the shoulder joint, formed by the coraco-acromial arch. The sub-deltoid bursa alone intervenes between the cushioned ball and the acromion, serving as the synovial membrane of the accessory socket, the space intervening when the parts are exposed in dissection being enough to admit only the end of the handle of the scalpel when pushed into it. Rupture of the long tendon of the biceps muscle might possibly cause some hitch in abducting the arm, but that secondary function of the tendon is exercised only when the biceps muscle is acting, and the former supposition that that rupture is a not infrequent occurrence is now well known to have arisen from a misinterpretation of the condition in which the tendon is usually found in cases of advanced rheumatoid disease.

The supposition that the rheumatic tendency has had the effect of delaying the union of the acromial epiphysis would imply that the rheumatic tendency had shown itself in early life, by the age of about the 25th year, and it is not evident why that tendency should single out the acromial epiphysis among all the epiphyses of the skeleton. The relation, if not due to injury causing fracture, must be sought in some operation of the advancing rheumatoid disease of the shoulder joint, effecting a separation of an already united epiphysis.

In the advanced stages of that disease there is great alteration of the parts, especially upwards, as well described by Adams. The capsular tendons are more or less completely absorbed, the upper part of the capsular ligament is more or less destroyed, allowing the head of the bone to pass upwards to contact with and friction against the coraco-acromial arch; the cartilage of the glenoid cavity and of the head of the humerus is more or less destroyed and replaced by eburnation, both socket and head are altered in shape and osteophytes and cartilaginous excrescences form; a large general socket results, embracing the altered old glenoid cavity and the supra-glenoid sub-acromial space, this capacious cavity embraced by a capsular ligament, the upper part

of which may be attached to the coraco-acromial arch, the lower part of it to beyond the anatomical neck of the altered humerus; and the cavity is filled with fluid and with synovial fringes and cartilaginous excrescences projecting into it. The long tendon of the biceps muscle may be in process of absorption but is generally gone within the joint and has acquired a new attachment to the humerus at the bicipital groove, from which that head of the muscle continues to exercise its function.

These extensive changes are well depicted by Adams in his Plate ii. and in Plate iii., fig. 2 (*op. cit.*, *b*). In regard to the upward passage of the humerus and its effect on the coraco-acromial arch he says (*loc. cit.*, *a*, p. 587):—

“Under the influence of the most usual form of this disease, all these parts intervening between the head of the humerus and the coraco-acromial arch or vault are absorbed; and the superior extremity of the head of the humerus at length comes into immediate contact with the concavity of the arch . . . its head being constantly pressed against the under surface or concavity of the coraco-acromial arch, not only do the processes of the scapula which form this arch at length show manifestly the effects of friction, but the upper portion of the acromial end of the clavicle does so equally. All these portions of bone are rendered concave, and are usually covered by a porcelain-like deposit, corresponding to an analogous polished surface which covers the convexity of the head of the humerus.

“In many cases in which the shoulder joint has long been the seat of this chronic disease, the *acromion process* has been found traversed in the line of junction of its epiphysis by a complete interruption of its continuity, as if fractured: we say as if fractured, for we are convinced that this solution of continuity of the acromion process is not really a fracture produced by violence, but a lesion, which so frequently exists in combination with chronic rheumatic arthritis of the shoulder, that we are compelled to look upon it, in these cases, as a peculiar organic change, the result of chronic rheumatic disease. We do not pretend to account for the separation of the acromion process into two portions; nor can we say why it is that the division usually occurs in the original line of the epiphysis, particularly at the late period of life at which we generally witness this phenomenon. In some of these cases we have found the acromion in a state of hypertrophy; in others in a state of atrophy; but in no case did there seem to be any attempt at ossific deposition on the contiguous surface of the separated portions of the acromion, a circumstance which might be expected if a fracture had occurred.”

As Adams says in *many* cases, it is worth while to inquire whether the association of separate acromion process with chronic rheumatoid disease is in reality a frequent occurrence. I have therefore made the following note of the number of such cases that have been recorded, so far as I am aware, noting at the same time whether there is evidence of the case having begun with an injury to the joint such as would account for fracture of the acromion process. An important point to note is, whether the acromion was separate in both shoulders. The condition of chronic rheumatoid disease, as Adams remarks, is frequently symmetrical, and the theory of fracture is not very likely to be applicable in cases of symmetrical separate acromion.

*Enumeration and particulars of cases in which separate acromion process was found associated with the condition of chronic rheumatoid disease of the shoulder joint.*

1. By Adams (*loc. cit.*, a, p. 590, fig. 429). Case of J. Byrne, male, æt. 55. Chronic rheumatoid disease of right shoulder for some years (no mention of left). When arm was raised humerus could be felt to strike against acromion. On post-mortem examination all the parts that lie normally between humerus and coraco-acromial arch had completely disappeared. Outer end of clavicle and the coracoid worn and excavated. *Acromion process* traversed from within outwards by a perfect solution of continuity, completely dividing it into two nearly equal portions, the two portions on same level, no ossific deposit at the separated edges. Same case figured in *loc. cit.*, b, Plate iii., fig. 2.

2. By Adams (*loc. cit.*, a, p. 588). Sex and side not mentioned. Specimen of advanced rheumatoid disease in Museum R.C.S. Dublin. Acromial end of clavicle unsupported, *acromion process* "removed" to extent of an inch, remainder of process thinner than natural and atrophied. This specimen shows that continued friction of the end of the humerus had led to absorption of the part of the acromion that is sometimes found separate.

3. In Sandifort's *Museum Anatomicum*, vol. iv., Table 25, fig. 2, referred to by Adams (*loc. cit.*, a, p. 599, fig. 431; also figured by Adams in *loc. cit.*, b, p. 163). Right side. Sandifort considered the condition of the parts to be the result of accident. Adams considers it a case simply of chronic rheumatic arthritis, and remarks—"The *acromion process* is divided into two portions; a phenomenon we have so frequently noticed to accompany this disease of the shoulder joint."

4. By R. W. Smith (*loc. cit.*, p. 12, figured in Pl. ii.). Side not mentioned and not evident from figure. "It was taken from the body of a woman of advanced age, who for many years suffered from the usual symptoms of this disease, and who, it was known, had never sustained any injury of the shoulder." As described, the changes on the soft parts and on the bones at the shoulder joint are the usual characters above noted of advanced chronic rheumatoid disease. "Upon removing the deltoid muscle the naked head of the humerus, elevated to the acromion process, came into view. . . . The *acromion*, about  $\frac{3}{4}$  inch from its extremity, was divided into two portions which were held together solely by the fibrous structure which invests the upper surface of the process; but this tissue was so stretched that the detached portion was separated from the remainder of the acromion by an interval of  $\frac{3}{4}$  inch. The under surface of the entire process was denuded of periosteum and covered with an ivory-like structure, which was also found investing the articular surfaces of the acromio-clavicular joint." The condition of the acromion in this case approaches that described in my Case No. 2, in which, however, there was no disease of the glenoid cavity.

5. By R. W. Smith (*loc. cit.*, p. 1, Pl. i., fig. 1). Male, æt. about 60. Case of *congenital* dislocation on dorsum of scapula in both shoulder joints, "in which chronic rheumatic disease had been established." Glenoid cavity situated on external aspect of neck, and no vestige of glenoid cavity in normal situation. Head of humerus "placed much further back than natural, and elevated so as to be in contact with the under surface of the acromion process." Disease much the same on both sides, but *acromion process* separate on right side only, on left side perfect. The separation of the right acromion is at about an inch from its anterior end; no deposition of bone along the line of separation; no displacement; the detached portion closely connected to the remainder of the process by fibrous tissue.

Here we have clearly a case of original malposition of the head of the humerus, serving for sixty years of life, in which there is at least no record of injury to account for the rheumatoid disease found in both shoulders. Although there was the "elevation to the acromion process," there apparently

was not naked contact of the head of the humerus with the acromion, as the tendons of the capsular muscles were perfect except that of the subscapularis, which had partially disappeared; and the capsular ligament was present, somewhat thicker than natural. The tendon of the biceps was present. There were osseous growths around the large glenoid cavity, and great changes on the form of the head of the humerus, with eburation. As the soft parts were present between the elevated humerus and the acromion, it would, on the whole, seem most reasonable to suppose that the separation of the right acromion had been due to some injury causing fracture.

6. By *R. W. Smith* (*loc. cit.*, p. 353). Owing to the loose way in which the cases are brought in, it is not at first evident that this was not the case No. 4 above noted, but on critical study it is seen to be a different case, and both sides are described. Female, age not given, "the patient laboured under the disease in both shoulder joints for several years before her death, but the affection had been established in the right articulation long before the left was attacked." Both sides show the changes in much advanced rheumatoid disease. The *acromion* separate on *both* sides, the detached portion at a considerable distance from the *basii-acromion*. On the *left* side—"The head of the humerus (with the intervention of the capsule) was pressed closely against the under surface of the acromion, which process, about  $\frac{3}{4}$  inch from its extremity, was divided into two portions separated fully  $\frac{1}{2}$  inch from each other, the fibrous structure which connected their upper surfaces having suffered a corresponding amount of elongation. The detached portion, along with the extremity of the clavicle, was also pushed upwards above the level of the remainder of the process."

On the *right* side, figured in Pl. iii., the changes were more advanced. "About  $\frac{3}{4}$  inch of the extremity of the *acromion* had altogether disappeared, and a broad, thick, concave plate of bone passed downwards from the shortened process, and became perfectly continuous with the upper and outer part of the margin of the glenoid cavity, which thus appeared as if a coracoid process had sprung from its external side. The acromial extremity of the clavicle was enlarged, excavated upon its under surface, and polished by the attrition of the head of the humerus; it was distant from the acromion fully  $\frac{3}{4}$  inch, but connected to it by an exceedingly dense fibrous tissue, which constituted the highest part of the capsule." The upper attachment of the capsule was transferred to the acromion, clavicle, and coracoid process, and was thin, "in many places as translucent as if it had been composed merely of synovial membrane." The tendons of the capsular muscles were flattened and expanded and detached from the humerus. The view here conveyed seems to be that the separate piece of the acromion had been floated down and become attached to the upper and outer edge of the glenoid cavity; and the author connects with this a reference to a preparation in the Dublin R.C.S. Museum, somewhat similar in other respects, of which Adams says "the acromial end of the clavicle is unsupported, and the acromion process has been removed for the amount of an inch in extent."

In this case it is seen that the capsule still intervened, but was very thin on the left side, allowing of close contact and pressure against the acromion, the side on which the detached acromion had suffered greatest displacement. Apart from the supposition of former injury to both shoulders, causing fracture, it appears not unreasonable to agree with Smith that the detachment of the acromions was a consequence of the ravages of the disease.

7. By *Mr John Gregory Smith* (*loc. cit.*, p. 280). Case in which the *acromion* was separate on *both* sides. Dissecting-room specimen, female, *æt.* 56. A short, stout, muscular subject, had worked at wash-tub up to time of sudden death. Appearances described are those of chronic rheumatoid disease of both shoulder joints, moderately advanced. No history to the case, but the author assumes "fracture." On *right* side—"There was an oblique fracture of the *acromion process* of the scapula, which had separated about an inch of its expanded extremity; it had not united by bone, but had formed an artificial joint

through the medium of cartilage, and was further strengthened by a fibro-ligamentous capsule." Sub-deltoid bursa communicated with cavity of shoulder joint by an irregular opening. Tendons of subscapularis and supraspinatus detached from their insertions and united to capsule. Tendon of biceps gone within joint. Under surface of acromion eburnated. On left side—"The *acromion process* of the scapula had been fractured precisely in the same situation as that of the opposite side, and formed a similar artificial joint." Sub-deltoid bursa much enlarged and thickened and communicated with shoulder joint. Tendons of subscapularis and supraspinatus partially detached from their insertions. Biceps tendon gone within joint, attached, as on right side, at bicipital groove.

Mr Gregory Smith's paper, 1834, is entitled "Pathological Appearances of seven cases of Injury of the Shoulder joint; with Remarks." The seven cases were in five subjects (two being in both shoulders) dissected in the Hunterian School of Anatomy. No previous history bearing on the nature of the cases, except that given in the one above related. His case 1 was a man, only left shoulder described; case 2, a woman *æt.* 30, only left side described; case 3, a woman *æt.* 38, only right side described; cases 4 and 5, that above given, with separate acromion on both sides; cases 6 and 7 (right and left shoulders of same subject), a man *æt.* 40. The appearances, well described, in all of them are those of the havoc wrought in the shoulder joint by chronic rheumatoid disease. The author assumes previous injury to the shoulder, apparently not acquainted with the characters presented in advanced rheumatoid disease, or with the question regarding separate acromion process. In only one of them (No. 7, above) was there separate acromion, but in one (his case 1) there was fracture of the outer end of the clavicle "which extended into its articulation with the acromion." These cases by J. G. Smith may be taken, simply, as well-marked cases of chronic rheumatoid disease, arising either from some supposed former injury, or, as held by Adams and R. W. Smith, as arising independently of injury; but in the one in which separate acromion occurred the precise symmetry of the separation is remarkable.

8, 9, 10, 11. Four of the specimens mentioned by Mr Lane (*loc. cit.*, *a*, p. 415 and p. 417). One (p. 415, side, sex and age not given) with great alteration of the head of the humerus and glenoid cavity. "There is also an ununited fracture of the *acromion*. Its direction is backwards and slightly outwards from a point a quarter of an inch internal to the clavicular facet." Adams and R. W. Smith would regard this case as one of advanced rheumatoid disease; Mr Lane regards the condition of the parts as "evidently produced by a fall on the shoulder." The fracture of the acromion, if fracture it was, is at a little behind the clavicular connexion, the usual situation in cases of separate acromion.

The other cases mentioned by Mr Lane (p. 417) are three of the five above referred to, given by him as "fair instances of ununited fracture of the acromion," in three of which there were "extensive rheumatoid changes in the shoulder joint." To one of these (the fourth of the five cases), as the separation was just behind the clavicle, the epiphysis theory would apply; but not to the first, in which the line of separation was just behind the front of the clavicular facet; nor to the third, in which the line was opposite the centre of the clavicular facet. These, therefore, are two cases in which separation of a part of the acromion co-existed with extensive rheumatoid changes, but in which fracture was the probable explanation, the injury that caused the fracture also setting up the rheumatoid condition.

12. The case No. 1, described by me in this paper. Subject *æt.* 80, case of old-standing sub-coracoid dislocation of the humerus with extensive rheumatoid disease of the shoulder joint (condition of latter noted fully in Appendix). Line of separation  $\frac{1}{2}$  inch in front of the posterior end of the clavicular facet. In this case the injury that caused the dislocation would readily account for a fracture of the acromion. The separation, however, is very close to the place at which it is usually found. My case No. 11, showing two separate ossicles,

might also be included in this list, as showing some little rheumatoid disease at the acromion, but the glenoid cavity is healthy.

*Interpretation of the preceding cases. Explanation of the co-existence.*

Adams and R. W. Smith are eager to prove that such cases are not cases of what various authors had described as partial dislocation upwards, arising from injury, in which the long tendon of the biceps had been ruptured; but are cases of chronic rheumatic arthritis, in which the displacement upwards was secondary, from the gradual destruction of the soft parts, leading to pressure of the humerus on the coraco-acromial arch and detachment of the acromion process. That position they may be said to have fully established as against the view of original traumatic dislocation upwards, with or from rupture of the biceps tendon, and as showing that extensive changes of the soft parts and of the bones are the result of long-standing rheumatoid disease. But they rather fail to satisfy that the rheumatoid condition may not have arisen, in at least some, perhaps in most, of their cases from an injury which at the same time had been enough to cause fracture of the acromion process, though not to cause dislocation of any kind. They have also been rather led away by the supposed frequency of the co-existence of separate acromion process and rheumatoid disease of the shoulder joint. Of the 12 cases above enumerated of that co-existence they had seen only the first six, and in addition could refer only to the seventh; and although they dwell on the occurrence of symmetry in the coincidence, the fact is that in only two of these seven cases (Nos. 6 and 7) was the acromion found to be separate in both shoulders. Considering the frequency of the occurrence of rheumatoid disease of the shoulder, their use of the term "many," as applied to the coincidence of separate acromion is, therefore, an exaggeration. Thus R. W. Smith, following Adams, says (*loc. cit.*, p. 355):—

"Among all the numerous and varied phenomena which occur during the progress of chronic rheumatic arthritis of the shoulder, there is none more remarkable, nor one for which it is more difficult to offer any satisfactory explanation, than the detachment of the extremity of the acromion process. It is most frequently to be noticed in the advanced stages of the disease, but I have more than once seen it at a period prior to the destruction of the tendon of the biceps; it is in many instances symmetrical, and in general occurs where in early life the epiphyses joined the remainder of the process. I have, however, in one instance found the entire of the acromion thus separated from the spine of the scapula. It may co-exist either with hypertrophy or atrophy of the acromion; it may occur with or without perforation of the capsular ligament; or with absorption in some instances, and displacement in others, of the tendon of the biceps, or finally in cases where the tendon is perfect as to structure and normal as to position."

In regard to the detachment of other epiphyses than that of the acromion, R. W. Smith says (*loc. cit.*, p. 356):—



“By those who have not made the subject of chronic rheumatic arthritis a special object of their study, it might be supposed that the singular solution of continuity of the osseous tissue, such as that to which I have been alluding, was only to be met with in the acromion process. This, however, is far from being the case, for I have seen half of an hypertrophied olecranon thus separated from the shaft of the ulna, in an aggravated case of this disease affecting the elbow joint, and in several instances of chronic rheumatic arthritis engaging the articulation of the knee, which are preserved in the Museum of the Richmond Hospital, large portions of the condyles and head of the tibia may be seen separated from the remainder of the bone. In one of these examples the detached mass is of such size as to embrace the insertion of the ligament of the patella.”

I leave it to surgical pathologists to say whether they find such separations of epiphyses a frequent occurrence. The following is R. W. Smith's recondite suggestion in the endeavour to account for separation of epiphyses taking place (*loc. cit.*, p. 357):—

“It would appear from the analytical investigations conducted by Mr Harper, and recorded by Mr Canton, that in this rheumatic disease of the shoulder a large proportion of the earthy matter naturally existing in the bone is removed; but I scarcely deem the knowledge of this fact adequate, of itself, to account for the solution of continuity in the acromion; and it appears to me that, in our endeavours to explain this remarkable phenomenon, we can at present only go so far as to suppose that, under the influence of this specific arthritic inflammation, the intimate structure of the bones undergoes some peculiar molecular alteration, the exact nature of which is as yet hidden from us, but the effects of which are to diminish its cohesive power, and to render it liable to yield to a pressure, which, though perhaps not powerful, is increasingly exerted upon it. In many of these cases I have found the affected bones soft, porous, and spongy: these conditions in some instances co-existing with increase of volume, constituting that state of the osseous tissue which Lobstein (*Traité d'Anatomie Pathologique*, tom. ii.) has described under the title of ‘Osteoporosis.’”

Mr Lane, in the second paper referred to (*loc. cit.*, b, 419), disposes summarily of the view of Adams and R. W. Smith, adopted by F. H. Hamilton, and repeated in English text-books of surgery—“That many of the specimens which have been regarded as ununited fractures of the acromion are really separations of the epiphysis”—holding that such cases have been the result of some injury causing fracture of the acromion, the injury afterwards setting up the rheumatoid condition.

In this thoughtful paper Mr Lane mentions that he is struck, as every one who has been long in dissecting-rooms must have been, with the great frequency with which so-called rheumatoid disease is seen, especially in old subjects. He regards the condition not as a “disease,” but as the result of pressure in over-use or disuse, influenced by occupation, or it may be lighted up by injury; modified by the vitality of the individual, the changes atrophic in feeble persons and by osteophytic growth or eburnation in powerfully-built hard-working men.

He says (p. 389), "I think I have succeeded in proving that most of the changes which are defined as indications of the presence of the so-called disease rheumatoid arthritis are purely physiological and in no way the product of any disease. They consist chiefly of what might be called accommodation changes."

Treating of "changes in the shoulder joint" in feeble old age (p. 437), Mr Lane speaks of gradual upward displacement of the humerus irrespective of rheumatoid changes. Partial atrophy of the supra-spinatus and of the upper parts of the other capsular muscles; extension of the articular surface of the humerus to the rotated-out and partially altered greater tuberosity; the ascent of the latter to the acromion, separated only by the muscles and capsule. The force that brings this about is, he says, that of the traction of the coraco-brachialis and biceps and deltoid muscles in the unused limb. That these are senile changes without rheumatoid appearances, but not often seen in the dissecting-room, as "the class from which our old subjects is drawn is one which is very much exposed to injury in every form." On this he concludes (p. 441):—

"I have now proved that the upward dislocation of the head of the humerus can and does take place without the presence of changes which are regarded as characteristic of rheumatic gout. I have also shown that injury to the shoulder-joint which has resulted in fracture of the acromion is always followed by the development of rheumatoid changes, and that these rheumatoid changes may be produced in the shoulder joint without any upward dislocation from injury to the shoulder in which the acromion has not been fractured."

Referring to the upward changes in the advance of the rheumatoid condition, Mr Lane says (p. 419):—

"As the head ascends, it rasps and destroys by its pressure the muscles that intervene between it and the under surface of the acromion, while they themselves tend to degenerate owing to the limited power of abduction in old age. Reaching the acromion the functional pressure exerted by the rough surface of the head of the humerus causes destructive changes in it, the periosteum being removed and the subjacent bone rubbed down and eburnated."

This clear description leaves us at the stage when, according to the separation theory of Adams, the detachment of the acromion should be effected, and it is not unreasonable to suppose that that detachment should be a further result of these "destructive changes" if it is the fact that the line of junction of the epiphysis is anatomically a weak point of the acromion. At the same time, on the supposition of fracture being the explanation, the question remains,—Why should the fracture be so often at that part corresponding to the place of junction of the epiphysis, and just behind the acromio-clavicular articulation?

## V. ANATOMICAL CONSIDERATIONS IN RELATION TO THE USUAL LOCALITY OF THE SEPARATION.

### (A.) STRENGTH OF THE ACROMION AT DIFFERENT PARTS.

If the under surface of the acromion is examined in a series of scapulæ it is seen that the *sub-acromial beam*, continued from the thick external border of the spine, spreads out as a smooth thickening upon about the posterior third of the acromion. The ridge varies a little in its sharpness, and in its distance from the posterior angle, the latter according to the degree of development of the posterior angle, but the expanded beam gives thickness and transverse convexity to the posterior third of the acromion, and the surface of that part is smooth. In front of this the inferior surface is concave in both directions and more or less foraminated, and the thickness is considerably less. The transverse concavity, generally present, is increased by the lip at the outer border to which the prolongation of the coraco-acromial ligament and the deepest part of the deltoid muscle are attached; and, internally, by the usually slightly projecting lip of the clavicular facet. The distinction between these two regions on the under surface of the acromion is on a line from the posterior end of the clavicular facet outwards and backwards to some way in front of the posterior angle of the acromion, the distance depending on the development of that angle, and the line marks off, stated generally, the posterior third of the acromion from what may be called the clavicular, or anti-clavicular, region of the acromion. The line of demarcation is not always striking to the eye, but is generally so, in some acromions very much so, owing to the change from the smooth convex region to the foraminated concave region.

On the *upper surface* the distinction strikes the eye less; the part corresponding to the foraminated region of the under surface is more foraminated than on the posterior third, but the foraminated area is more encroached on by the raised smooth outer part for the deltoid attachment than on the under surface.

When the thickness of the acromion is taken at where the sub-acromial beam has spread into the general thickening, and at where the foraminated region has begun, the thinning at the latter is found to be considerable. An average thickness of 9 millimeters at the former has fallen to an average of 7 millimeters, a diminution of about  $\frac{1}{2}$  inch. This line of demarcation corresponds to where the epiphysis has united, and if there is such a thing as detachment of an already united epiphysis from the ravages of advanced rheumatoid arthritis, it is where we might expect the acromion to give way. On the other hand, if the numerous museum specimens of separate acromion are to be interpreted as ununited fractures, we have no less an explanation

of the fracture being so often at that locality. Anatomically it is the weak point of the acromion.

In regard to the possible detachment of this part of the acromion in advancing rheumatoid disease, if the epiphysis has not yet united, the disintegration of the intervening layer of cartilage might be expected, but the rheumatoid condition is generally one belonging to advanced life. Due time being allowed for the consolidating process, here, as elsewhere, no traces remain of former distinctness; the transition of the cancellous architecture is gradual. In the variously cut sections of the acromion in my collection there is no definite difference in the internal structure at or near the place of union. The enclosing lamina of dense bone above has become very thin opposite the clavicular facet; that below, rising as a thick lamina from the sub-acromial beam, is more marked than the upper and is prolonged somewhat further than the upper lamina. The areolæ of the cancellous tissue are smaller and more rounded in the anterior than in the posterior half of the acromion, and in the latter than in the spine, but there is no abrupt change at the post-clavicular line. Any weakness of the acromion here is to be sought, not in traces of the consolidation of the epiphysis, but in the above noted thinning of the acromion at this part. This thinness would account for the acromion giving way in advancing rheumatoid disintegration here rather than further back, but not for its giving way at the post-clavicular region more than in any part of the clavicular region, along which the thinness of the acromion continues to be as great.

#### (B.) INFLUENCE OF THE CLAVICULAR CONNEXION IN DETERMINING THE LOCALITY OF SEPARATE ACROMION.

The connexion with the clavicle appears to me to be the most important consideration in endeavouring to explain why the locality of the separation is usually at the post-clavicular line and also why the separation is usually permanent; and that whether the cause is fracture, or epiphyseal non-union, or detachment by rheumatoid disintegration. This part of the acromion is bound to the clavicle and is supported by it; while just behind this, where the support ceases, the acromion is thinner than it is a little further back. For these two reasons combined the post-clavicular line may be regarded as the weak point of the acromion. Then, when the separation is once established, the pushings of the clavicle against the acromion, as may be seen by a glance at Fig. 1 (page 9), must tend to prevent non-union and render the separation permanent.

While, as noticed above, fracture of the acromion may occur occasionally at any part of the process, the fact is that in all of the 13 specimens described in this paper, and they are not picked cases, the line of separation is at or close to the posterior end of

the clavicular facet. They have all a family likeness, and for this the anatomical reason just given appears to account satisfactorily.

Of the 13 specimens, the clavicular facet was entirely on the separate acromial ossicle in 8, the line of separation just behind the facet. Figs. 6 and 6a (p. 9), show this most usual position of the separation just behind the facet, and symmetrically so, although one ossicle, the left, is longer than the other. In one (No. 7, fig. 8) the line of separation is about  $\frac{1}{8}$  inch behind the facet; in one (No. 12)  $\frac{3}{12}$  inch behind a very short ( $\frac{6}{12}$  inch) facet. In one (No. 2) the atrophied ossicle was loosely connected, but there was no connexion of the clavicle to the basi-acromion. The position of the joint a little in front of the hinder end of the facet, occurring in four of the specimens, is more interesting, as giving the clavicle a rest on the basi-acromion. In two of these (Nos. 10 and 13)  $\frac{1}{12}$  inch of the facet was on the basi-acromion, the facets, respectively, of  $\frac{1}{12}$  and  $\frac{8}{12}$  inch in length.

In one (No. 6, fig. 7) about a fourth part of a  $\frac{9}{12}$  inch long facet, and in No. 1 a fourth part of an  $\frac{8}{12}$  inch long facet, were on the basi-acromion. As the latter was in one of the dissections, I was able to test the effect of that on the movement of the ossicle by the clavicle. When the clavicle was pushed outwards the ossicle was freely moved, when the clavicle was pushed outwards and backwards the movement was arrested against the basi-acromion. Thus, in the ordinary movements of the shoulder, the fact of a small part of the facet being on the basi-acromion does not prevent the clavicle from causing movement of the ossicle, and thus causing the false joint in the case of fracture or maintaining a separate acromion from whatever cause arising. The connexion of the clavicle to the basi-acromion by the posterior part of the acromio-clavicular ligaments, present in all the dissections except in case No. 2, did not prevent the movement of the ossicle, free enough to be recognisable in the living body on careful examination.

Applying these considerations to the case of *fracture*, we see that, in whatever way the force comes, the anterior part of the acromion is supported by the clavicle, the force taking effect at the post-clavicular line. A stroke from above by a limited weapon will break the acromion at the part struck; but when struck broadly, or when the force is transverse as in a fall on the shoulder, it is manifest that the natural part for the giving way to take place is at the post-clavicular line. If fracture is to be accepted as the interpretation of the numerous specimens of separate acromion seen in the dissecting-room and in museums, we have thus a fair explanation of the separation being usually at or close to the post-clavicular line, and a good reason for the separation being rendered permanent by the constant pushings of the clavicle against the detached part of the acromion.

It appears reasonable to regard the clavicular connexion as having an influence also in relation to the *non-union of the epiphysis*. The post-clavicular line corresponds to the line of meeting of the epiphysis with the basi-acromion. Indeed, the position and extent of the epiphysis may be regarded as being in adaptation to the clavicular connexion. In young persons, up to about the 25th year, the same kind of injury that produces fracture in the adult consolidated acromion is still more likely to fracture the intervening layer of cartilage, and the movements of the parts will

maintain the separation and lead to the formation of a diarthrodial joint, as in fracture through the bony tissue of the adult. That, of course, is "fracture" (diastasis), but facilitated by the position of the epiphyseal line.

But apart from injury, the connexion to the clavicle might be regarded as a possible cause of non-union of the epiphysis, in the constant transverse force in the abutting of the clavicle against the epiphysis; that either in persons of feeble constitution or in young persons of exceptional activity, and the same cause would continue the separation. Apart from the continual operation of forces causing movement, we see a familiar instance of non-union continuing through life, if it passes the usual period of union, in the permanence of the suture between the right and left frontal bones of man; and in those mammals in which that is the normal condition we see that when forces operate, as in those of them that have horns, the suture becomes dentated. The influence of movement from natural forces in converting anywhere a synarthrodial into a diarthrodial joint is not to be overlooked. The theory of delayed union and naturally perpetuated non-union might be employed to account for separate acromion being symmetrical; but that is a rare occurrence, while, if the theory were well founded, separate acromion ought to be a very frequent occurrence, either symmetrically, or, if on one side only, much more frequently on the right side than on the left.

The supposed influence of *rheumatoid disease* in causing separate acromion must be taken to operate differently, according as it occurs before or after the union of the epiphysis. If before union, the rheumatic tendency might be supposed to delay the union, thereafter perpetuated by the same tendency or by the movements of the parts, and it should be symmetrical; but the rheumatic condition is comparatively rare in early life. Were advancing rheumatoid disease to find the epiphysis still not united, it might be supposed to attack first the intervening cartilage and thus detach the acromion, but advanced rheumatoid disease is a condition belonging to middle life or old age. If after consolidation, from the excavation of the acromion in the advanced stage of rheumatoid arthritis, the support given by the clavicular connexion should, perhaps, render the post-clavicular line the readiest part to suffer, being the place also where the forward thinning of the acromion has begun. The argument for this cause, that rheumatoid disease of the shoulder joint is often symmetrical, and that this accounts for symmetrical separate acromion, is met by my case No. 3, in which there was no rheumatoid disease, and by the fact that only two cases of the co-existence have been recorded. As against this theory, too, there is the consideration that advanced rheumatoid disease of the shoulder joint is of frequent occurrence but that the co-existence of separate acromion is exceptional. Considering that rheumatoid disease often follows on some injury,

as we see so often in old-standing cases of unreduced dislocation of the humerus, one cannot avoid the suspicion that the occasional co-existence of separate acromion with rheumatoid disease of the shoulder joint has been due to some injury which had fractured the acromion and led on to the rheumatoid condition.

## VI. GENERAL CONSIDERATIONS.

### (A.) RELATION TO SEX, AGE, AND SIDE.

In regard to *sex* we would expect fracture, and also rheumatoid disease, to be most frequent in the male, but the above cases, so far as certain, do not show that preponderance. Among my 13 cases, of the 4 dissections in which the sex had been noted, 3 were female (Nos. 2, 3, and 4), 1 male (No. 5). Of the 8 dried specimens, 4 seem from their robustness to have been males, the other 4 uncertain. Of the 6 cases of Adams, R. W. Smith, J. G. Smith, and that in the Sir Charles Bell collection above noted, 3 were in males, 3 in females.<sup>1</sup>

<sup>1</sup> Statistics relating to sex and age are liable to be influenced by a variety of circumstances. Thus, of the two schools in which I have taught anatomy; in one, the female subjects preponderated, the ratio of female to male being as about 3 to 2; while, in the other, the proportion was nearly the reverse; so that, in a total of 1200 of which I have record of the sex, the sexes came to be almost exactly equal. A more interesting statistic, as showing the basis for dissecting-room observations, is that giving the ages as well as the sex. For this I can give a statistic of only 890, as in some the precise age was not known and for about 240 the record of the age is not now available. I give the ages in five-year periods. It is seen that the number between the ages of 5 and 15 is small; that the number is large between the ages of 55 and 75, giving ample ground for advanced rheumatoid arthritis; and that after the age of 65, females preponderate, very much so after the age of 75.

Ages inclusive.	Total.	Male.	Female.	Ages inclusive.	Total.	Male.	Female.
1 to 5	37	18	19				
6 " 10	3	1	2	51 to 55	432	213	219
11 " 15	7	3	4	56 " 60	52	30	22
16 " 20	32	14	18	61 " 65	80	41	39
21 " 25	56	36	20	66 " 70	72	41	31
26 " 30	73	29	44	66 " 70	86	38	48
31 " 35	46	22	24	71 " 75	70	33	37
36 " 40	63	30	33	76 " 80	54	18	36
41 " 45	46	23	23	81 " 85	28	8	20
46 " 50	69	37	32	86 " 90	11	2	9
				91 " 95	5	1	4
	432	213	219		890	425	465*

\* This apparent preponderance of females is owing to my not now having the ages of those in the earlier years in the school in which males preponderated. Of the 1200 of which I have record of the sex, the sexes are almost exactly balanced.

In regard to proportions of sex derived from *hospital* statistics it is to be borne in mind that the male patients are more numerous than the females in

As regards *age*, when noted, it is seen in Table II. that, of my 13 cases, the first four had reached the 80th year, three of them females; the fifth case was *æt.* 64, male; Adam's case (*loc. cit.*, a, p. 590), *æt.* 55, male; R. W. Smith's case (*loc. cit.*, p. 1), *æt.* 60, male; and J. G. Smith's case, *æt.* 56, female. It is to be kept in mind that a considerable proportion of the subjects in the dissecting-room are elderly or old, and, in regard to the first four of these cases, that among the aged subjects females preponderate.

I find no distinct record of a case of separate acromion occurring in a young person at the place of union with the epiphysis. Reference is made above, from Mr Lane, to the ages of the ten cases of fracture of the acromion recognised and treated at the Middlesex Hospital in the ten years ending 1879; one within the first five years of life, one between 15 and 30, four between 30 and 45; the remaining four above 45 years of age. But no definite information is given as to the locality of the fracture.

*Side.*—Reference is made above to the fact that of the 14 cases given by me 3 were on the right side, 9 on the left side, 1 on both sides, and 1 certainly on the right side, doubtfully on the left. Adding to these the cases quoted (Mr Lane, 5 right, 1 left; other authors, 3 right, 2 on both sides), we have a total of 11 on the right side, 9 on the left side, 3 on both sides, and 1 doubtfully on both sides, but certainly on the right. That is a much less preponderance on the right side than we would expect on the fracture theory, when the greater exposure of the right limb to accidents

general hospitals, even in the physicians' wards. In our great hospital here the proportion is as 4 to 3, and in the hospital of the other school in which I taught the predominance of the male patients was in about the same proportion.

Looking to the general proportion of the sexes, the statistics of the Registrar-General for Scotland, for the year 1895, show in regard to births that for every 100 females there are 105·2 males, but that after about the age of 10 years females preponderate in the population, the disproportion increasing as years go on, the ranks of the males thinned by emigration, the army, the sea, and greater exposure to accident and the causes of disease. Although in the total deaths females preponderate (total in Scotland, in 1895, 81,864, males 40,726, females 41,138), the proportion is reversed in the case of deaths in the large towns (males 5511, females 5382.) Dr Blair Cunynghame, of the Registrar-General's Office, Edinburgh, has kindly given me the following figures showing the proportion of women to men in the two towns to which my anatomical statistics refer. In Edinburgh, with a total population of 261,225 (census of 1891) there were 22,715 more females than males, being a ratio of 119·05 females to every 100 males. In Aberdeen, with a total population of 124,943, there were 9803 more females than males, being a ratio of 117·03 females to every 100 males. In Edinburgh, in a total of 27,554 persons at the working age of 20, the excess of women was 2872; at the age of 25 the excess was 3065. As to the sexes at old age, at the age of 70, the numbers in Edinburgh were, men 1170, women 2174; at the age of 80, men 278, women 640; at the age of 90, men 21, women 53. In Aberdeen, at the age of 70, there were 692 men, 1264 women; at the age of 80, 180 men, 415 women; at the age of 90, 7 men, 32 women. These figures are not without interest in relation to the anatomical statistic above given.



is considered. Thus in the account of 41 "Specimens of Complete Dislocation of the Humerus preserved in the Anatomical Museums of London," given by Sir W. H. Flower (*loc. cit., infra*), in 36 of which the side is noted, 25 were of the right side, 11 of the left.<sup>1</sup>

#### (B.) CONCLUSIONS.

1. Fracture of the acromion process is, in all probability, a much more frequent occurrence than is usually supposed. It is liable to be overlooked from the absence, generally, of displacement, but may be detected on careful manipulation by the movement of the fragment and by crepitus. This conclusion appears to be fully established by the researches of Mr Arbutnot Lane.

2. The fracture may occur at any part; in front of the clavicular facet; opposite the facet; or behind the facet. Just behind the facet appears to be the usual locality.

3. This post-clavicular line is what may be termed the weak point of the acromion. This for two reasons: (*a*) The acromion is thick behind, supported and strengthened by the sub-acromial beam of the spine, and becomes thinner just behind the clavicular facet: (*b*) And, more especially, as the acromion in front of this is bound to and supported by the clavicle. Forces therefore tend to tell most on this part of the acromion.

4. The alleged relation between *rheumatoid arthritis* of the shoulder joint and separate acromion, as dwelt on by Adams and R. W. Smith, appears to be founded on misapprehension. The cases in which the co-existence has been recorded are not numerous, only 7 in number among the numerous cases of that disease in its advanced condition in which the parts were dissected. While there need be no difficulty in accepting the opinion that among the upward ravages seen in chronic rheumatic disease of the shoulder joint detachment of an ununited epiphysis, or detachment of an ossified acromion, may be one of the disintegrations, there is reason to believe that the rheumatoid condition is usually the result of former injury to the shoulder by which fracture of the acromion had been caused.

5. In regard to the non-union or separation of the *epiphysis* theory, the following are the considerations for and against it:— The place of junction of the epiphysis corresponds to the post-clavicular line. But although the epiphyseal line thus corresponds to the weak point of the acromion it is not the cause of the weakness, as after the union is completed (between the 22nd and 25th

<sup>1</sup> Although in the above-noted 24 cases of separate acromion only 3 are recorded as having the condition on both sides, it is not to be absolutely concluded that it may not have been present on the other side also in some of the other cases. Although it may, perhaps, be inferred generally that the condition was present only on the side preserved for the museums, those who are familiar with dissecting-room work will not infer that the other side was always examined or notes made, but we must go by what is known.

year) there is no difference in the internal structure and no special thinness exactly at the line of union. The correspondence of the two lines is incidental except in so far that the extent of the epiphysis appears to be in adaptation to the clavicular connexion. But the fact of the correspondence of the two lines introduces the element of doubt in the interpretation as between fracture and epiphyseal separation, when the separation occurs, as it usually does, at the post-clavicular line.

*In support* of the epiphysis theory may be given: (a.) That the usual place of separation corresponds to the place of epiphyseal meeting, expressed generally. Looking to the figures 2 to 5 of the development of the acromion given in the Plate (p. 9) we appear to have a satisfactory explanation of the locality being the usual one. (b.) It is conceivable that union may be delayed beyond the 25th year, and that if union does not occur at the usual period non-union may be permanent, as seen occasionally in the case of the inter-frontal suture. (c.) If the specimen has been from a subject under the 25th year the intervening layer of cartilage may have been broken by an accident to the shoulder, and the synarthrodial connexion thus converted into a diarthrodial joint by the movement of the parts, as after fracture of the ossified acromion followed by non-union. That, of course, brings the case into the category of fracture (diastasis), but the line is determined by the epiphysis.

*Against* the theories of non-union or detachment of the epiphysis, as occurring in the living body, may be put: (a.) There is a source of fallacy in regard to the interpretation of some specimens met with, in that they are but normal scapulæ just under the age at which the acromial epiphysis (the last of the epiphyses of the scapula to unite) is consolidated; the separation having taken place during maceration. Such scapulæ, wanting the epi-acromion but otherwise full-grown, are to be seen in museums. But in regard to specimens undoubtedly beyond that age—(b.) The line of post-clavicular separation is not exactly what would be expected had the cause been non-union or detachment of the epiphysis. Among the 13 specimens described in Part III., the separation begins, on the inner side, in some exactly at or very close to the posterior end of the facet (figs. 6 and 6a, case 3, and in cases 4, 5, 8, 9, and 11); in some a little in front of that point (fig. 8, case 7,  $\frac{1}{8}$  inch in front; case 12,  $\frac{3}{12}$  inch in front); in some a little behind (fig. 7, case 6,  $\frac{1}{6}$  inch behind; case 1,  $\frac{1}{8}$  inch behind; cases 10 and 13,  $\frac{1}{12}$  inch behind).

These moderate variations in the starting-point of the separation on the inner side do not, perhaps, go against the epiphysis theory, as we do not know that normally the posterior limits of the epiphysis and of the facet correspond precisely to each other, but the outward course of the line of separation is not much, in some the opposite, of what we should expect when we bear in mind that

epiphyses have a very definite shape. As seen in figs. 2 to 5, the epiphysis meets the basi-acromion in a line curving outwards and very much backwards from the posterior end of the clavicular facet. Even should the posterior nucleus (\*fig. 4) not unite with the main body of the epiphysis, the line of union is still curved with the concavity backwards. But in some of my specimens, as described, there is very little backward direction of the outer part of the line; in some the outward direction is even a little forwards. Figs. 6 and 6a show the direction to be different on the two sides in the same person, though symmetrical in regard to beginning on the inside just behind the facet. On the whole, the general direction of the line of separation, while somewhat undulating, may be regarded as transverse or nearly so.

Allowance must, no doubt, be made for any changes of form during perhaps many years of active working at a false joint after a supposed epiphyseal detachment; but, on the whole, when the line of separation usually seen is considered, the epiphyseal theory, whether by delayed union or by detachment, fails to satisfy, while the line of separation, as usually seen, with its minor variations at the post-clavicular region and its general transverse direction, tends to support the fracture theory.

6. The occasional occurrence of *symmetrical* separate acromion appears at first to be a difficulty in accepting the view that in all cases the condition is one of fracture, while it is intelligible on the theory of delayed union of the epiphysis, or as a result of advanced rheumatoid disease of both shoulder joints. Of the three cases above referred to, in two (those by R. W. Smith and J. G. Smith) there was rheumatoid disease, while in my case (No. 3, a female *æt.* 82, figs. 6 and 6a) there was no disease. These two theories, however, when critically examined as above, must be regarded as improbable, as inapplicable to at least the great majority of the specimens. It remains only to inquire whether the seemingly not very likely occurrence of fracture of both acromions can be believed as, after all, not so very unlikely to occur. This difficulty will disappear when we think of the occasional occurrence of dislocation of both shoulder joints, a result implying a much more forcible cause than would suffice to fracture the acromion. Of this occurrence I subjoin a reference to five cases.<sup>1</sup>

<sup>1</sup> 1. Adult male, the case in the Sir Charles Bell collection in the Edinburgh College of Surgeons' Museum, above noticed. Old-standing unreduced sub-glenoid dislocation of the humerus of both sides. Rheumatoid changes in both shoulder joints. Acromion fractured on left side with bony union.

2. In the same museum (New Cat., p. 193, Nos. 5, 6). Plaster cast of the front of the chest and shoulders of an adult. The dislocation on the right side was recent, that on the left side was old standing. Presented by the late Dr J. D. Gillespie, surgeon to the Royal Infirmary of Edinburgh. The dislocation is very manifest in both shoulders.

3. Case recorded by Mr C. E. Oldacres, of Daventry (*British Medical*

With such examples of symmetrical injury, in two of them both sides dislocated simultaneously, we need have little difficulty in believing in the occurrence of symmetrical fracture of the acromion process; the less so when we think of the number of tumbles the old frequenters of the drink-shops of London, Dublin, and Edinburgh must have had, first on one shoulder then on the other, before arriving finally at the dissecting-room.

The epiphysis theory is attractive to the anatomical mind, and it is not easy to part with the impression of one's earlier years in the dissecting-room, but when the evidence is critically examined has to be abandoned for the fracture theory. The explanation above given of the frequency of the locality in which the fracture is found to have occurred, the post-clavicular line, giving the family likeness to the great majority of the specimens, is, however, no less an anatomical one, and appears to me to be the true interpretation.

#### APPENDIX.

(a.) *Condition of the parts in Case No. 1, considered in relation to the occurrence of indentation or fracture at the anatomical neck of the humerus, accompanying sub-coracoid dislocation.*

Besides presenting characters of advanced rheumatoid arthritis following on the old injury, the chief interest of this specimen is in the femur-like elongation of the neck of the humerus, with a deep and wide post-cervical excavation. This character seems to render the specimen worthy of exact examination in relation to the apparently not infrequent occurrence of injury to the humerus as a complication in sub-coracoid dislocation. The condition in this specimen resembles that in the specimen noticed by me in 1862 (*Edinburgh Medical Journal*, vol. viii., 1862, p. 274) from a male subject *æt.* 76, now in the museum of the Edinburgh College of Surgeons (*New Cat.*, vol i., p. 192, No. 5, 5) to which reference will be made below in comparison.

*Condition of the parts.* Subscapularis muscle present and of good size for a rather undersized scapula. *Ligaments:* Good capsular ligament all round. *Scapular attachments:* above, to inner half of anterior border of coracoid, for 1 inch, the coracoid broadened anteriorly at this part; from this, attachment

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*Journal*, Nov. 9, 1895, p. 1162). A very muscular man; fell from roof of cottage; sub-glenoid dislocation of both shoulder joints, and fracture of left thigh-bone. Dislocations reduced.

4. Dr R. M'Dongall, of Gladstone, South Australia, relates (*same Journal*, 8th February 1896, p. 383) that the late Professor Sir George Macleod showed his pupils, at the Glasgow Royal Infirmary, in 1871, a case of dislocation into the axilla in both shoulders, and their reduction, in a woman *æt.* 45 or 50.

5. By Professor T. P. Anderson, of Sydney (*same Journal*, June 6, 1896), p. 1385). In the dissecting-room; old-standing dislocation of both shoulder joints. The man had been able to go about his work, as a drayman, until he was accidentally killed when intoxicated.

continued internally and below along inner and lower edge of new glenoid cavity. Externally and below, attachment normal along posterior and lower border of old glenoid cavity. *Attachments to humerus*: outer part to outer edge of great tuberosity; anterior and posterior parts to anterior and posterior borders of elongated neck; inner part, normally along original anatomical neck. Outer part of capsule thicker than normal, inner part not thickened. Thus, the capsule embraces both glenoid cavities above, and, below, not only the head but the elongated neck, out to the outer border of the great tuberosity.

*Interior of the joint*: Shows old-standing sub-coracoid dislocation, new sub-coracoid glenoid cavity, great elongation of neck of humerus, with deep and broad excavation behind, and changes from rheumatoid disease on both surfaces of joint, but these not extending upwards above old glenoid cavity. *Old glenoid cavity*: dimensions normal, height  $1\frac{1}{2}$  inches, breadth 1 inch; spongy at middle for half its breadth, upon which soft part of inner wall of great tuberosity of humerus has rested; eburnated behind, before and below the soft spongy part. Inner edge projects more at upper than lower half (the reverse of the normal); eburnated area towards this most projecting part corresponds to eburnated area on inner wall of great tuberosity, the projecting edge itself sinking to the bottom of the post-cervical excavation on humerus. Less projecting lower half of glenoid edge is rather rounded off towards new glenoid cavity, but is not much worn down.

*New glenoid cavity*: On subscapular fossa immediately to inner side of old cavity and continuous with it round the projecting eburnated inter-glenoid ridge. Dimensions greater than of old cavity, vertically 2 inches, transversely  $1\frac{3}{4}$  at lower half, less than that at upper half. It reaches up on base of coracoid process for  $\frac{2}{3}$  inch, occupying entire breadth of coracoid, which is here flattened and excavated vertically to form part of cavity. This coracoid part of new cavity reaches higher up than old cavity by about  $\frac{1}{4}$  inch. Inner boundary of new cavity below this is marked by a sharp prominent ridge, running downwards and outwards on subscapular fossa. Concavity of new cavity much more marked vertically than transversely, owing to vertical curve of coracoid part. Surface of new cavity, spongy, with fibrous tufts, along inner half, eburnated along outer half. Against the eburnated part has played the back part of head of humerus, the part of the rheumatoid ring marked off from the smooth central part of the head. The projecting eburnated inter-glenoid ridge forms a bluntly acute angle, is above middle of old cavity, and opposite about middle of new cavity.

*Humerus*.—On articular surface of head two areas to be distinguished,—a central ovoid area, size of pulp of thumb, smooth but thin articular cartilage which can be sliced with the knife; around this central area is a ring of irregularly nodulated smooth eburnated surface. The central area plays against inner (now anterior) side of capsular ligament, behind subscapularis muscle, bulging these forwards. The only part of the eburnated ring that has not rested merely on capsular ligament is the back and upper part, where it is increased in breadth, to about an inch, outwards to the excavation on the neck; this is the part that has played in the upper and inner part of the new glenoid cavity. This gives back of head a flattened shape (with a little transverse convexity) for an inch in length outwards. The normal anatomical neck should intersect the middle of this flattened part, but the form has been altered by the elongation at the back of the head throwing the anatomical neck outwards to where the excavation begins, in adaptation to the new glenoid cavity.

The *post-cervical excavation*,  $1\frac{1}{2}$  inch wide at the opening, about  $\frac{2}{3}$  inch deep, receives a good-sized finger. Outer wall eburnated on its upper part, where it played against the eburnated part on upper half of old glenoid cavity. Bottom of excavation over  $\frac{2}{3}$  inch in length, has received the prominent wedge-like inter-glenoid ridge, and is smooth and polished behind, corresponding to lower part of the ridge. Inner wall of excavation, which has

rested in lower and outer part of new glenoid cavity, is less smoothly eburnated.

*Divergence of great tuberosity.* The great tuberosity is abnormally inclined outwards from line of shaft, here to extent of  $\frac{3}{4}$  inch, the inclination beginning abruptly at  $1\frac{1}{4}$  inch from summit of tuberosity. (Normal inclination in robust humeri only  $\frac{1}{4}$  inch, or less, and much more gradual.) *Appearances of former fracture.* Below great tuberosity, on outer surface of shaft, there is appearance as if a scale or splinter of bone,  $1\frac{1}{2}$  inch in length,  $\frac{5}{8}$  in breadth, lower end  $2\frac{3}{4}$  inches from summit of tuberosity, had been broken off and united. Scale is continuous with the tuberosity, as if both had been broken off together; the grooves bounding it are continued behind from just behind posterior border of tuberosity, in front from neck of humerus some way in front of tuberosity. The grooves are sharp and very evident, less so towards tuberosity. That there had been fracture is confirmed on making a longitudinal section of the head, neck and upper  $\frac{1}{2}$  of the shaft. The appearances then seen are:—Medullary canal of shaft reaches into outer half of neck. Cancellous tissue of head easily broken down. Line of articular lamina of head distinctly seen to extend underneath the eburnated raised ring above noticed, which rises upon the lamina for  $\frac{1}{4}$  inch. Line of former fracture well seen as a narrow lamina of dense bone intersecting the cancellous tissue internal and external to it; begins above at outer part of neck,  $\frac{3}{4}$  inch to 1 inch from head, extends down for  $2\frac{1}{4}$  inches, as indicated on outer surface of shaft, where it unites with dense wall of medullary canal. This clearly establishes that there had been fracture accompanying the sub-coracoid dislocation, the fracture splitting off the greater tuberosity and a long splinter of the shaft. The anatomical neck, internal to the fracture, had then in the course of time become elongated in adaptation to the movements on the inter-glenoid ridge and the new glenoid cavity.

[In the 1862 specimen, above referred to, the abnormal divergence of the tuberosity is less, but is very marked, begins about  $1\frac{1}{4}$  inch from the summit of the tuberosity, and at that locality there is a jagged line towards the outside and behind as if this portion had been broken off with the tuberosity and united; but this indication is not nearly so distinct as in the new specimen. The post-cervical excavation is even wider and deeper. The description above given of the altered form of the head and of the two glenoid cavities might serve for both specimens, except that in the 1862 specimen the new glenoid cavity does not reach up on the coracoid, its upper end being  $\frac{1}{2}$  inch lower than the upper end of the old glenoid cavity. The neck of the humerus, though greatly elongated, femur-like, is not so oblique as in the new specimen, so that top of the great tuberosity is not much below level of top of head. The long tendon of the biceps was normal.]

The parts show further results of chronic *rheumatoid arthritis*: various separate ossifications projecting into the joint from the capsular ligament or in the ligament. (a.) At inner and lower part of cavity, two, one size of small almond, one size of pea, hanging into the joint from the capsule near inner edge of new glenoid cavity. (b.) From upper part of capsule, two, size of pea. (c.) At bottom of post-cervical excavation, attached by fibrous tissue, one, larger than pea. (d.) At front of excavation, in the capsular ligament, two, a large one, 1 inch by  $\frac{1}{4}$  inch, and one larger than pea. Many tufts and fibrous laminae hang into the joint at where the capsular ligament is attached. The rheumatoid changes have not extended upwards towards the acromion, not above the old glenoid cavity. The attachment of the *biceps tendon* has been transferred to the humerus, firmly attached below the normally placed lesser tuberosity. The tendon is now about half the normal bulk and not tubular. (See my note on this condition of the transplanted tendon in the *Edinburgh Medical Journal*, vol. i., 1856, p. 953.) A vestige of the upper part of the tendon is seen partly incorporated with the capsule.

*Movements and adaptations.* The movements of the shoulder joint permitted by the ligaments are, flexion and extension pretty free, abduction and

adduction very moderate; not rotation, being prevented by the ligaments and by the elongated form of the neck. When the parts at the joint are placed in their acquired adaptation and the scapula held naturally, the shaft of the humerus is directed very much outwards. When the humerus is placed naturally, with only a little outward direction, the base of the scapula slopes very much outwards and upwards. It would seem that this oblique position of the scapula must have been acquired as an adaptation to the movements of the shoulder joint after the dislocation. The new adaptations at the shoulder joint may be stated generally as, that the back of the head rests in the new glenoid cavity, retained there by the subscapularis muscle and the untorn capsule; that the great tuberosity rests by its inner wall in the old glenoid cavity; and that the projecting inter-glenoid ridge sinks into the post-cervical excavation and forms a kind of pivot on which the two prominences move in their respective sockets.

*Altered form and proportions of the upper end of the humerus.* The increase in the length of the neck is seen by the following measurements:—Total length from outer wall of great tuberosity to middle of end of head,  $2\frac{5}{8}$  inches. (The same measurement in a normal muscular humerus, 2 inches.) The  $2\frac{5}{8}$  inches are apportioned thus: head with broadening externally, carrying the lesser tuberosity,  $1\frac{1}{8}$  inch; post-cervical excavation, at bottom,  $\frac{7}{8}$  inch; great tuberosity at same level,  $\frac{5}{8}$  inch. The neck is very oblique as well as elongated, so that the great tuberosity is considerably below the level of the head, to the extent of  $1\frac{1}{4}$  inch in the natural adaptation, with the shaft of the humerus inclined outwards; to the extent of  $1\frac{1}{2}$  inch when the humerus is held vertically. (In the normal humerus, about  $\frac{1}{4}$  inch.)

The fracture which appears to have accompanied the dislocation has been at the outer side of the part of the anatomical neck opposite the great tuberosity, and thence carried down so as to detach the great tuberosity with a splinter of the shaft below it.

(b.) It is a question of interest whether the post-cervical excavation, or groove, often seen in specimens of old unreduced sub-coracoid dislocation, is brought about in the course of time by the pressure of the inter-glenoid ridge in adaptation to the new position of the parts, or has had its beginning in an indentation or fracture caused by the impinging of the prominent inner glenoid margin against the humerus at the time of the dislocation. Various writings bearing on this question may be referred to.<sup>1</sup>

*Dr Joseph Bell* points out the exact relation of the humerus to the scapula in the sub-coracoid and sub-glenoid dislocations, in the usual cases of moderate dislocation in which only the front of the capsule is torn, and the capsular

<sup>1</sup> *Sir William H. Flower*—"On the Pathological Changes produced in the Shoulder Joint by Traumatic Dislocation, as derived from an Examination of all the Specimens illustrating this Injury in the Museums of London," *Trans. Path. Soc. London*, 1861. *Dr Joseph Bell*—"On the Nomenclature of Scapulo-Humeral Dislocations," *Edin. Med. Journal*, May 1863. *Mr Frederick S. Eve*—"A Case of Sub-coracoid Dislocation of the Humerus, with the formation of an Indentation on the posterior surface of the Head, the joint being unopened; with Remarks on the Mode of Production of Fracture of the Anatomical Neck, with Dislocation," *Med. Chir. Trans. London*, March 1880. *Professor E. H. Bennett*, of Dublin, *Brit. Med. Journal*, August 1880, p. 349—"On Fracture of the Neck of the Humerus, as a complication of Dislocation of the Shoulder." *Mr Francis M. Caird*—"The Shoulder Joint in relation to certain Dislocations and Fractures," *Edin. Med. Journal*, Nov. 1886.

muscles not torn but stretched so as to retain the humerus in its dislocated position. That, in the sub-coracoid form, the projecting anterior glenoid border is received into the part of the anatomical neck behind the greater tuberosity, which he terms "the posterior groove of the humerus"; and that, in the sub-glenoid form, the projecting posterior ridge of the axillary border, in like manner, projects into and may be retained in the groove of the humerus. He further explains that this catching of the glenoid margin in the posterior groove of the humerus may go the length of producing "consecutive fissure of the head of the humerus," separating the greater tuberosity, caused "by the sharp edge of the glenoid border being forced as a wedge against the posterior groove of the humerus."

In the case related by *Mr Eve*,—that of a man, æt. 36, knocked down by a train while working on the line, the head of the bone distinctly felt beneath the coracoid process and the dislocation easily reduced, death twelve hours after the accident,—“on the posterior surface of the head of the humerus, at the margin of the articular cartilage, there was a deep vertical indentation or groove, into which the anterior margin of the glenoid cavity accurately fitted;” and he remarks—“I conclude that the groove was produced by the forcible impact of the humerus against the anterior margin of the glenoid cavity.”

In this interesting paper *Mr Eve* says—“There are two dried specimens of shoulder joints in the museum at St Bartholomew’s Hospital, showing dislocation of the humerus forwards with the formation of a groove or trochlear surface on the posterior portion of the head, evidently produced by attrition against the anterior margin of the glenoid cavity, which has itself been considerably worn away. *Malgaigne* (*Fractures et Luxations*, p. 496) has noticed the occasional occurrence of grooves on the head of the humerus after dislocation. He mentions two cases described by *Sédillot*, which presented much the same appearances as the above. In these specimens, as in the two former, the furrows were hollowed out by the movements of the head upon the glenoid margin, but *Malgaigne* remarks (p. 497) that he thinks it is a question if they are not sometimes produced, ‘at the moment of dislocation, by the crushing of the head of the humerus upon the border of the glenoid cavity,’ a conjecture which is proved correct by the case related. It is also probable that the commencement of the groove might, in some of the specimens mentioned, have been formed in a similar manner.”

*Mr Eve* goes on to suggest that the occurrence of such a groove “may explain the mode of production of fracture of the anatomical neck with dislocation of the head of the humerus forwards. If the blow had been sufficiently forcible the head of the humerus would probably have been chipped off by the anterior margin of the glenoid cavity, instead of simply an indentation being produced by it.” The specimen is in St Bartholomew’s Hospital Museum; *Cat.*, vol. i., p. 146, No. 1019.

*Prof. Bennett* “exhibited five examples of dislocation of the shoulder, complicated by fracture of the upper extremity of the humerus. In one of these, the dislocation was recent and the fracture incomplete; in the remaining, the fractures were completely united.” Having reviewed the opinions of *Delpech*, *Cooper*, *Malgaigne*, *Smith*, and, lastly, of *Mr Eve*, on the mechanism of the double lesion, the author advanced his views, founded on the examination of the series of recorded cases and on the specimens exhibited, and expressed them in the following conclusions:—“1. Fracture of the upper extremity of the humerus occurring as a complication of the dislocation commences at that part of the anatomical neck which rests, after dislocation has taken place, against the border of the glenoid cavity. 2. It is caused by pressure of the humerus against the sharp edge of the glenoid cavity, probably the result of a constrained position, preventing the separation of the elbow from the side as in ordinary dislocations. 3. While the fracture starts at the anatomical neck, and may follow it strictly, it commonly passes into the shaft detaching the lesser tuberosity with the head. 4. The fracture is neither comminuted nor impacted.” In the *Dublin Medical Journal* of March 1884, p. 359, *Prof.*



Bennett notices a specimen of Fracture of upper extremity of Humerus, history unknown, which had in his opinion been consecutive to dislocation; agreeing with his former specimens and confirming his opinion. The head united by ligaments to both scapula and humerus.

Mr Caird describes and figures two specimens of sub-coracoid dislocation in the museum of Edinburgh University. In one of these, a recent dislocation, there is an indented fracture, beginning at the upper and back part of the anatomical neck, forming a groove  $\frac{1}{4}$  inch deep,  $\frac{1}{2}$  inch broad, and  $1\frac{1}{2}$  inches long, into which "the anterior lip of the glenoid cavity accurately fits." In the other, a dried specimen, the indentation is in the head of the humerus, 1 inch long,  $\frac{1}{2}$  inch deep, and the indentation "evidently corresponds to the anterior edge of the glenoid."

Mr Caird finds that "We can produce, although with some difficulty, and in a very artificial manner, a similar injury on the cadaver. Make a sub-coracoid dislocation, lay the subject prone, and strike the scapula violently. It will be found that a series of lesions may be obtained, varying in degree from a mere bruise of the glenoid cartilage onwards to indentation of the head of the humerus or to complete intracapsular fracture, the anterior lip of the glenoid, hard and dense, cutting into the cancellated tissue of the humerus like a knife." Reviewing the cases and opinions of the authors above quoted, he expresses the opinion—"It would appear, therefore, that we are justified in recognising the true type of sub-coracoid dislocation as being *always* associated with an indentation fracture of the head of the humerus caused by the dense, hard, anterior lip of the glenoid."

Sir William Flower's paper (*loc. cit.*) is of great value from the large number of facts he records and from the conclusions founded upon them. He fully recognises, writing in 1861, the misinterpretation of Adams in considering the rheumatoid condition to precede instead of being consequent on the dislocation. He brings out the striking fact that, of the 41 specimens of traumatic dislocation noted by him in the London museums, the position is *sub-coracoid* in 32—"placed upon the anterior margin of the glenoid fossa, or neck of the scapula, *immediately beneath* the coracoid process." The dislocation was backwards in four. In one (No. 11) the position had been described as "sub-clavicular," but the dislocation was recent and the head of the humerus had been removed from that position in putting up the preparation. In several other specimens described in the catalogues as of that kind, "the new socket is distinctly seen in the preparation to be immediately below the coracoid process. So that there is no specimen really illustrating the "sub-clavicular" variety, as defined by Sir A. Cooper ('the head of the os humeri placed below the middle of the clavicle, and on the sternal side of the coracoid process'), which is said by some authors to be second in order of frequency." In two of the specimens (Nos. 6 and 38) the position was "sub-glenoid"; as compared with the sub-coracoid, "somewhat lower down, the new socket being formed partly at the expense of the lower and anterior portion of the glenoid fossa, and partly on the anterior edge of the inferior costa of the scapula, the upper part of the head of the humerus being at a distance of somewhat less than an inch below the coracoid process. In one specimen (No. 23), the position is intermediate between this and the first mentioned (sub-coracoid) form. In no case is the head of the humerus placed entirely below the glenoid cavity."

As bearing on the great *preponderance of the sub-coracoid position* in museum specimens, Mr Caird (*loc. cit.*) remarks—"From the fact that our museums contain many examples of dislocation forwards, we are apt to imagine that the downward displacement is much rarer. One must remember, however, that this may mean no more than a relative difficulty in reduction. Of nine *recent* cases admitted lately to the Royal Infirmary, only one was sub-coracoid, eight were clearly sub-glenoid." On the other hand, Sir William Flower says, in regard to the sub-coracoid preponderance and in regard to the theory that the head of the humerus in neglected sub-glenoid dislocations passes up

to the sub-coracoid position—"As the great frequency of sub-coracoid dislocation observed in this series does not accord with the descriptions of this injury generally given in the standard surgical works of this country, and might lead to the supposition, that in these examples of neglected dislocation, the head of the humerus had in the process of time assumed a position which did not at first belong to it, I should mention, that in upwards of fifty cases recently observed in living patients, in a very large majority it could be distinctly felt immediately below the coracoid process, and that this has already been recognised as the typical position by most surgeons of the modern French school." These two statistical statements are not reconcilable. A more extended statistic is required. If Mr Caird is right, we are led to the conclusion either that sub-coracoid dislocations are frequently overlooked, which is not very probable, or that they are often accompanied by fracture of the humerus, rendering reduction difficult or impossible.

In regard to the occurrence of a *post-cervical groove* on the humerus, and its condition, in the 32 specimens of sub-coracoid dislocation tabulated by Sir W. Flower, the following appears:—Described as "grooved," 7; as "slightly grooved," 4; as "deeply grooved," 5; the groove generally at or near the outer side between head and great tuberosity. In two of "recent" dislocation, head of humerus "unchanged," but in one of them part of great tuberosity torn off. In two "rather recent," one "apparently unchanged"; the other "slightly grooved between head and great tuberosity." Two showed fracture at the anatomical neck, one of them with fibrous union, the other with false joint. One "apparently unchanged." The others variously altered in shape, but not described as grooved. Thus, in about half of the specimens of sub-coracoid dislocation noted by Sir W. Flower, the humerus showed the groove in various degrees. As the size of the groove is not given, it does not appear whether it amounted to the wide and deep excavation seen in my two specimens.

In regard to the *position of the new glenoid cavity* the distinction is made between the *sub-coracoid* proper and the *intra-coracoid* of Malgaigne (not "sub-clavicular"), in that in the latter the new cavity is quite internal to the old cavity, without grooving of the humerus; while in the former the new cavity encroaches on the old cavity, with grooving of the humerus. The distinction is but one of degree. According to that distinction the dislocation in my two specimens would be rather *intra-coracoid*, as the new cavity encroaches very little on the old one; but the position is strictly *sub-coracoid* in both. This may be co-related to the great width of the *post-cervical* excavation in them, with consequent elongation of the neck, enabling the head to play in the new cavity and the tuberosity to play against the old cavity.

In regard to the *causation of the post-cervical groove or excavation*, it is a question whether it is the result of gradual adaptation, the glenoid edge resting originally in the natural groove presented by the part of the anatomical neck at the great tuberosity and working a deeper groove in the course of time. Supposing that to be the position of parts at the dislocation, and so retained by the capsular muscles, there need be no difficulty in receiving that view of the formation of a deeper and broader groove in the humerus. More probably there is, to begin with, an indentation or partial fracture at or near the anatomical neck, caused by the impact of the glenoid edge. In at least one of my two specimens (case No. 1) there had evidently been a fracture, splitting off the greater tuberosity with a splinter of the shaft; bony union had taken place, and the great elongation of the neck must have been a gradual process in adaptation to the movement on the prominent inter-glenoid ridge.

(c.) *Note on the normal neck of the humerus.* The part loosely, though conveniently in surgery, called the "surgical neck," is not definable except generally as about the upper inch or so of the shaft, where it enlarges towards the head and tuberosities, as seen on all sides. In diastasis the line of fracture is quite at the upper part of the surgical neck, as the line of the epiphysis, transverse with minor undulations, cuts off exactly the articular head and the tuberosities. But

most of the so-called surgical neck belongs on the inner side to the anatomical neck, or neck proper of the humerus. The "anatomical neck" appears to be understood by some as if passing round on all sides just beyond the articular head. At the tuberosities it is a well-marked furrow,  $\frac{1}{6}$  to  $\frac{1}{4}$  inch in breadth, but below the inner half of the head the bone slants uniformly towards the shaft for about  $\frac{3}{4}$  inch, without any definite limit below, or any above except the edge of the cartilage, or, in the macerated bone, the articular lamina. The length and obliquity of the neck may be observed equally in front and back views. Seen in front, the axis of the shaft runs up at the outer edge of the lesser tuberosity; the axis of the neck intersects the middle of the lesser tuberosity, and runs out below the greater tuberosity about  $1\frac{1}{2}$  inches from its summit. Seen behind, the axis of the shaft runs up a little internal to the great tuberosity; the axis of the neck passes at the lower edge of the great tuberosity. A considerably obtuse angle is formed below where the axes of the shaft and neck meet. The direction of the neck is inwards, upwards, and backwards; the backward direction best seen on viewing the bone from the outer side.

In dissection, it is seen that most of the neck is within the capsule. At the tuberosities the capsular tendons, there representing the capsular ligament, are attached to the smooth facets of the tuberosities, encroaching a little inwards at the outer side of the anatomical neck, but the narrow anatomical neck is seen free and covered by synovial membrane. Below the head, the capsular ligament is attached from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch from the articular edge, leaving that extent of periosteal bone within the capsule and covered by synovial membrane.

It may here be observed, in regard to possible impact against the glenoid edge, that, in manipulating with a normal unopened capsule, it does not appear possible to bring the posterior part of the anatomical neck against the anterior glenoid edge by any movement or position without rupture of the front of the capsule. By extreme rotation inwards the fore part of the anatomical neck may be brought in relation with the anterior glenoid edge; by extreme rotation outwards the back of the anatomical neck may be brought in relation with the no less projecting posterior glenoid edge. By gliding movement forwards or backwards, without or with rotation, the articular surface of the head may be brought against the anterior or the posterior glenoid edge and might be indented or fissured vertically; by downward gliding the articular surface may be brought against the lower end of the glenoid margin, and thus produce a transverse fissure. All without any tearing of the capsular ligament.

In regard to the sub-glenoid position the following may be noted. The sub-glenoid or axillary groove, extending along about the upper half of the axillary border, is at the upper part  $\frac{1}{2}$  inch in breadth and faces obliquely forwards. Here, its outer border, the upper part of the true axillary border, is prominent and rough, where it attaches the long head of the triceps, and this ridge might catch in the back of the anatomical neck in a sub-glenoid dislocation. The beam forming the inner boundary of the groove, stronger below than the outer boundary, falls at about an inch from the glenoid cavity, and thus leaves a vertically concave area where the back of the head of the humerus may rest in a sub-glenoid dislocation.

The specimens described in this paper were shown and considered anatomically at the Anatomical Society of Great Britain and Ireland, in London, in Nov. 1887, and shown and considered surgically at the Edinburgh Medico-Chirurgical Society in June 1894.

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*Mr Joseph Bell* said that he thought Prof. Struthers had scored a very distinct point by bringing out how common it was that the

terminal portion of the acromion only had separated. He thought that fracture of the acromion was not common. It was common, however, to have fracture of the external end of the clavicle. Thinking over the cases he had seen, he could recall one in which he thought that he had not succeeded in curing a fracture of the acromion, but the patient was not a penny the worse. No doubt it was a case of separation of the terminal end of the acromion.

*Dr Ronaldson* said that he had seen two cases in father and child of separate acromion process on both sides. He believed that when it was congenital it would always be found on both sides.

*Mr David Wallace* said that the subject was one that had interested him and others in the surgical department at the University for some time. Prof. Chiene happened to possess at least three, if not four specimens of the condition. It had been pointed out for a long while that fracture of the acromion process was rare, and it had always been referred to in the books as one of the instances where a persistent epiphysis might simulate fracture. Arbutnot Lane said that, examining in the dissecting-rooms for fracture, he had found that fractures of the acromion process were the most common in the body. They were probably really examples of the condition to which Prof. Struthers had alluded—persistent epiphysis. With regard to the diagnosis between fracture of the acromion and persistent epiphysis, in the former they would get crepitus. Further, as Hamilton pointed out, in fracture of the acromion they would have displacement downwards of the fractured portion by the fibres of the deltoid attached to it. In the specimens in the Surgical Museum there was evidence of chronic rheumatoid arthritis, but this was due probably simply to the too free movement of the head of the humerus upon the under surface of the acromion process, because the osteophytic projection and the so-called porcellanous deposit, which was really simply a burnishing of the bone, was chiefly situated on the under surface. The complete destruction of the long tendon of the biceps to which Prof. Struthers had referred was a further marked evidence in support of this view. Mr Wallace had seen two such cases.

# APPENDIX II.

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## ON A CASE OF DIABETIC NEURITIS,

WITH A

## DESCRIPTION OF THE POST-MORTEM EXAMINATION OF THE NERVES AND MUSCLES.

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AND

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*(Communicated to the Society, 15th May 1895.)*

## ON A CASE OF DIABETIC NEURITIS, ETC.

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SYMPTOMS indicating involvement of the nervous system in diabetes mellitus have been recognised for several years, especially since Bouchard drew attention, in 1884, to the loss of the knee-jerk in this disease. More recently, it has been definitely shown, by the occurrence of such symptoms as motor weakness, paræsthesiæ, neuralgic pain, and impairment of vision, that peripheral neuritis may be associated with this disease. The evidence of this association, however, has mainly been derived from clinical observation, and in only a few instances—such as a case published by Pryce,<sup>1</sup> two cases published by Eichhorst,<sup>2</sup> and three cases published by Auché<sup>3</sup>—has it been demonstrated by post-mortem examination that the symptoms indicating peripheral neuritis had actually been produced by this condition. It may, therefore, serve a useful purpose to put on record an additional case, in which post-mortem examination confirmed the diagnosis of neuritis in a patient suffering from diabetes mellitus, more especially as certain changes were found in the muscles, which appear to have hitherto been overlooked.

William S., aged 36, an engine-fitter, was admitted to Ward 26 of the Royal Infirmary on 12th October 1894, suffering from feebleness, emaciation, thirst, polyuria and amblyopia. No tendency to any special disease was indicated in his family history. There was no indication of syphilis. His circumstances had been fairly good, and he had been temperate as regards alcohol, rarely drinking whisky or beer, and then only on occasional Saturdays, when he never exceeded two glasses of whisky. Until August 1893 he had enjoyed excellent health. Several weeks before this time he had suffered much anxiety on account of the severe illness of his wife, and had also been deprived of sufficient sleep and rest. Gradually, thirst and increase in the frequency of micturition attracted his attention, and, with advancing weakness, caused him to enter the Western Infirmary of Glasgow in September 1893, when it was discovered that he was suffering from diabetes mellitus. He improved greatly under treatment, mainly

<sup>1</sup> *Brain*, vol. xvi. 1893, p. 416.

<sup>2</sup> *Archiv für Path. Anat. und Physiolog.*, vols. cxxvii. and cxxviii.

<sup>3</sup> *Archives de Médecine Expérimentale et d'Anatomie pathologique*, 1890.

dietetic, and was able to return home in nine weeks. Before he resumed work, however, his wife again became ill, and renewed anxieties and discomforts caused a relapse in his condition. His vision also became impaired, especially for near objects, and in June 1894 he found himself unable to read.

When admitted into the Royal Infirmary, he weighed only 8 st. 2 lbs., his weight formerly having been 10 st.; he had a careworn expression, subnormal temperature and dry skin, with two pigmented areas on the right side of the abdomen. When on ordinary diet his appetite was great, he had much thirst, and his bowels were constipated. Neither the liver nor spleen was enlarged, and although the skin was pale in colour, examination of the blood showed that there was no anæmia.<sup>1</sup> The heart's action was feeble, and there were phthisical changes in the upper lobe of each lung.

On his admission, and so long as he was on ordinary diet, from the 13th to the 25th of October, the urine gave the following averages:—

Quantity in twenty-four hours, 195·4 ounces; specific gravity, 1038·8; grains of urea per diem, 568·8; grains of glucose per diem, 7187.

Under restricted diet, the averages from the 26th to the 31st October were—quantity in twenty-four hours, 132·5 ounces; specific gravity, 1035·4; grains of urea per diem, 738; grains of glucose per diem, 3862.

When to the restricted diet pancreatin was added, from 1st November to 21st November 1894 the averages were—quantity in twenty-four hours, 120·3 ounces; specific gravity, 1030·9; grains of urea per diem, 657·9; grains of glucose per diem, 2736.

No albumen was found in the urine during the patient's residence in the Infirmary.

In addition to these ordinary symptoms of diabetes mellitus, *the patient, from the first day of his admission, suffered from pain in the calves of the leg and in the ankles, of a dull cramp-like character, which was felt when he stood after walking a short distance, and also frequently while he was in bed; and he stated that he had suffered from these pains for three or four months previous to his admission. They varied in severity, and were sometimes so severe as to waken him from sleep.* There was, however, no numbness, formication, or abnormal sensation of heat and cold.

It was ascertained that the tactile, thermal, and pain sensibilities were normal; *but both calves were found to be very tender even on slight pressure, and pressure also caused pain when applied to the front of the ankles, over the tibia and fibula of each leg, to the external lateral ligament of the right ankle, to the anterior tibial*

<sup>1</sup> The red corpuscles numbered 4,960,000, and the leucocytes 13,000 per c. mm.; there was 74 per cent. of hæmoglobin present, and the specific gravity was 1062.

*muscles of the right leg, to the left inner hamstring muscles, and to the right biceps.*

The knee-jerk was absent on both sides, and there was no exaggeration of any of the superficial or deep reflexes.

The patient could stand without swaying with the feet close together and the eyes shut; no peculiarity was observable in his gait when walking, and the sense of position of the limbs was normal.

Amblyopia was present, and small objects, such as the fingers, could not be seen when at a greater distance than 4 feet. There was a central scotoma for red in each eye, but the field of vision was not restricted, nor were the fundi abnormal. There was no defect in any of the other special senses.

Notwithstanding the improvement in the urine, and a slight increase in body-weight, the progress of the illness was unfavourable. Mental torpor, general weakness, gastric disorder, and cardiac weakness gradually increased, until at the middle of October the patient could no longer leave his bed. On the 19th of October it was observed that the chloroform or acetone-like odour, which had for some time been present in the breath, had become much more pronounced. The symptoms of pulmonary disease became more urgent, vision more defective, and the pain and tenderness of the legs more severe. On the 20th of November the breathing was rapid, dyspnœa appeared and soon passed into orthopnœa, the pulse became rapid and weak, and in a few hours the patient was unconscious. Death occurred on the forenoon of the following day; but no albumen appeared in the urine, nor could fat be detected in the blood, when a specimen of the blood under the microscope was tested with osmic acid. While the patient was in the hospital, and until three days before his death, the temperature had usually been subnormal, and had always been so on each morning, ranging between 96°·4 and 98° F. On the 18th of November the morning temperature, however, was 99° F., and on the 21st of November it was 99°·4 F.

At the post-mortem examination, made on the 22nd of November, it was found that both lungs were phthisical, with breaking down of limited areas of lung substance, but no other gross pathological changes were observed.

The optic and posterior tibial nerves and portions of the tibialis posticus muscle were examined histologically after being carefully hardened in Müller's fluid in the usual way. The optic nerve (see Fig. 1) showed well-marked parenchymatous degeneration in various stages. Sections stained by the Weigert-Pal method showed a somewhat reniform area, a little to one side of the centre of the nerve, in which the degeneration was most advanced. This was evidenced by the pallor of this portion. The great majority of the fibres failed altogether to stain, or only held the colour very lightly. Round this was a zone in which the stain



was much deeper, but even here the fibres were not by any means all normal. The periphery of the nerve showed a stage of degeneration intermediate between that present in the two parts already described. Pieces of the nerve from a part closely adjacent to that from which these sections were cut were transferred directly from Müller's fluid, in which they were originally hardened, into Marchi's fluid (Müller's fluid 2 parts, osmic acid 1 per cent. sol., 1 part) for ten days, and were then embedded in celloidin and cut in the ordinary manner. The black droplets of the osmic acid reduced by the degenerating myeline were most marked, not, as one might have expected, in the pale reniform area, but at one extremity of this and in the darkly-stained zone in its concavity. A number of black droplets were also found, but to a less marked extent, in the narrow peripheral zone.

A comparison of the conditions revealed by those two methods of staining showed that the degeneration was most marked in the paler part of the central area, where the myeline had become almost entirely absorbed, and that it was still proceeding actively immediately around this area, as was shown by the fact that the fibres still retained the hæmatoxylin, although in an abnormal way, as well as the osmic acid.

Staining by ordinary hæmatoxylin and eosin showed some slight increase in the connective tissue in the endoneurium and perineurium. The cells were mostly rounded or spindle-shaped, and were apparently most marked in the portion of the nerve which was most degenerated. Here they were found between the individual nerve fibres and in the connective tissue binding groups of fibres together, but their number was so small compared with the degree of degeneration that they could only be looked upon as being formed secondarily. The posterior tibial nerve, examined in longitudinal sections stained by Pal's method, showed very high degrees of degeneration, as is represented in Fig. 2. The degeneration was apparently almost entirely limited to the myeline sheath of the fibres, the continuity of the axis-cylinder being maintained in almost every individual instance. The axis-cylinder in most of the fibres, whatever their stage of degeneration, showed varicosities which varied in breadth and in length in different instances. (See specially A, C, D, E, Fig. 2.) The myeline showed very different stages of degeneration in different fibres. In some (A and G) the axis-cylinder was almost entirely denuded. In others (such as F) the degeneration was beginning, apparently by a splitting up of the myeline into short segments. These left the axis-cylinder uncovered here and there, and also showed numerous annular constrictions which retained the hæmatoxylin somewhat more intensely. In other sections the myeline seems to have broken down into smaller masses and granules, which stained lightly with hæmatoxylin. The sheath of

Schwann seemed to persist after the disappearance of the myeline. In most of the fibres it was shown, still retaining the dark hæmatoxylin stain, with apparent interruptions where it had been completely decolorised. In sections stained with alum-hæmatoxylin and eosin the persistent portions of the myeline sheath presented an imbricated or funnel-shaped appearance (this is seen to some extent in fibres C and H) which was probably due to the remains of the Schmidt-Lanternmann's constrictions. In some of the less degenerated fibres there was an irregular network which stained with eosin. The nuclei of the sheath of Schwann were increased in size and formed marked fusiform swellings on alternate sides of the axis-cylinders of those fibres from which the medullary sheath had disappeared.

Small portions of the tibialis posticus muscle were cut in celloidin, some of them after previous treatment with Marchi's fluid. Sections stained by Pal's method showed a degeneration in the interfascicular branches of the nerve which was of the same nature as that already described as occurring in its trunk. Sections stained by the ordinary hæmatoxylin and eosin method showed here and there a slight increase in the distinctness of the longitudinal striation, but no change in the nuclei of the sarcolemma, or of the connective tissue between the fibres. The longitudinal striation was explained by the reaction of the osmic acid, and was seen to be due to *rows of fine fat granules between the fibrillæ of the muscle. The granules were all extremely minute, and seemed to be developed from the cement substance rather than the muscle fibrils.* In the portions of the muscle where this degeneration was found the *transverse striation of the fibres had disappeared.* This degeneration did not affect each muscle fibre or the whole of any one fibre, but rather short lengths of the fibres, and gave the section an appearance somewhat like that seen in fatty degeneration of the heart (Fig. 3). The change was quite different from that found in a degenerating muscle after section of the nerve.

It was somewhat surprising that the optic disc and retina showed to the ophthalmoscope so little evidence of any morbid change. This may have been due to the nature of the degeneration, which appeared to be the same as that found in the posterior tibial nerve.

From the above description it is evident that the nerves examined were affected with that peculiar form of degeneration which is usually (and somewhat inappropriately) termed parenchymatous neuritis.

In this case the special features were the degeneration of the medullary sheath, the persistence of the axis-cylinder, and the absence of secondary degeneration in the nerves or in their end organs (retina, muscle).

The process seems to be most nearly allied to that first discovered

and described by Gombault (*Archives de Neurologie*, parts 1 and 2, 1880) as *néurite segmentaire periaxiale*. It was found by Gombault in the nerves of guinea-pigs to which minute quantities of white lead had been administered along with their food for long periods, that even before symptoms indicating any nerve lesion had appeared the peripheral nerves became altered in the following manner:—The medullary sheaths over short lengths of the fibre, (corresponding to one or more nodes of Ranvier), between which the fibre remained healthy, showed a breaking-up of the medullary sheath into fine granules. These granules became ultimately absorbed either in solution or by the intervention of leucocytes, which had made their way through the primitive sheath of Schwann. The axis-cylinder, however, persisted, although never in a quite normal condition, being always more or less varicose or moniliform, and generally showing a more distinct longitudinal striation than in the normal condition. This twofold feature of persistence of axis-cylinder and degeneration of the medullary sheath is indicated by Gombault's term, *néurite segmentaire periaxiale*.

It is true that in the nerves examined the segmentary character of the change in the medullary sheath was not present, or at least was not made out, as in all cases the myeline had degenerated over a longitudinal extent corresponding to a large number of nodes of Ranvier, without any indication of healthy intervening portions of fibre; but in other respects—the breaking down of the myeline, and the persistence and irregular swellings of the axis-cylinder—the process closely resembled Gombault's neuritis.

In this respect it differs from most cases of alcoholic neuritis, where the axis-cylinders usually give way and secondary degeneration follows in the muscles. It is, of course, possible that this change might take place also in severe cases, or in late stages of diabetic neuritis.

The amount of newly-formed connective tissue was slight, and not such as to suggest any primary interstitial neuritis. (This would appear to have been present in Pryce's case.) When this occurs as a secondary change in parenchymatous neuritis it would appear to vary greatly in amount in different cases, being generally well marked in alcoholic neuritis, and, on the other hand, absent in neuritis due to mercury.

In this case it was much more marked in the optic than in the posterior tibial nerve. This may be attributable partly to the fact that the neuritis in the optic nerve antedated that in the latter nerve; but also to the fact that the patient was a heavy smoker, and that the neuritis in the optic nerve may have been partly due to tobacco.

The change in the muscle is a somewhat remarkable one, which we have not had occasion to examine before, and cannot find any account of in the literature of peripheral neuritis. It is character-

ized by small areas in which fat droplets form in rows between the fibrillæ of the muscle. This might be termed a *disseminated interfibrillary fatty degeneration of the muscle*, and is accompanied by a gradual disappearance of the transverse striation of the muscle fibre, but not by any multiplication of the nuclei either of the sarcolemma or of the connective tissue. This process is so completely different from that which results from descending degeneration of a nerve that it appears to be due to an independent and direct action on the muscle itself of some toxic substance rather than to any want of trophic influence on the part of the nerve.

The pathological changes and the associated symptoms seem clearly to be caused by a toxic substance, which acts directly on the medullary sheath in the nerve fibres, although recent examinations of the spinal cord in analogous processes render it probable that the nerve cells are also involved. The localized affection of the myeline of the peripheral nerves shows a curious similarity to the affection of the fibres in the central nervous system in multiple sclerosis, where the myeline is also first affected while the axis-cylinders long remain unaltered, and, consequently, secondary degeneration appears only occasionally. There the process always begins in the neighbourhood of some vessel, and appears due to the exudation from it of some poisonous substance capable of directly acting on the hyaline sheath. In both cases the affection of the nerve is more a degenerative than an inflammatory one.

The actual causal relationship between these pathological changes and diabetes mellitus has not yet been ascertained. In the greater number of the recorded cases, the age of the patients has been above 50. As in the neuritis originating from other causes, much diversity occurs in the symptoms of original cases. Leyden has distinguished three groups:—1. *The hyperæsthetic or neuralgic group*, in which pain is the prominent symptom, although it is usually accompanied with weakness in the parts supplied by the affected nerves; 2. *The motor or paralytic group*, in which paralysis predominates, implicating especially the muscles of the lower extremities, and accompanied with loss of the knee-jerks; 3. *The ataxic group*, in which, however, the ataxia is nearly always complicated with paræsthesia and with loss of the tendon-reflexes, but in which the iris-reflexes are retained.

Marked symptoms of neuritis appear to occur in only a small number of cases of diabetes mellitus, although slight symptoms are probably present in a considerable proportion of the cases. The amount of sugar in the urine does not appear to have much significance. Cases of diabetic neuritis have occurred both where the quantity of sugar was large and where only a small quantity was present in the urine. Further, some experiments made by Auché seem to prove that sugar itself, even under prolonged contact, is unable to produce neuritis, or, indeed, of acting more

injuriously on nerves than water alone. The neuritis is apparently caused by a substance originating from sugar, and not improbably from a derivative of alcohol produced from the glucose of diabetes mellitus.

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*Dr Bruce*, in explanation of the specimens, said that the nerves available were optics, and posterior tibial, muscle from gastrocnemii, and the spinal cord was also removed, but he had not had an opportunity of examining it yet. In the clinical description the central scotoma for red was noted, but no change in optic discs was revealed on ophthalmoscopic examination. On microscopic examination of the nerves they found in one of the optic nerves, apparently also in the other, a very marked area of degeneration in the centre. Central part pale, and dark fibres at the periphery. Round droplets and varicose swellings, well seen in some of the other specimens under the microscope, were not a recent degeneration, *i.e.*, had lasted for a month or six weeks. The medullary sheath had become segmented. After a time the droplets were removed and the change was not seen. The method of preparation was that of Marchi, an Italian observer. The specimens were put in Muller's fluid for a month, then passed into Muller's fluid and osmic acid for ten days or a fortnight. Wherever a fibre was degenerate there were rows of black droplets; healthy fibre showed a grey colour. The two sets, stained by Pal's method and by Marchi's method, put it beyond doubt that there had been marked neuritis in two stages,—an older part, in the centre, of more advanced atrophy, and around that a somewhat more recent part. Both posterior tibial nerves were examined, and the degeneration was found to be more marked in the right than in the left. It was present both in the trunk and in the minute interior muscular branches. The changes in muscle were somewhat different from any he had ever had an opportunity of examining before, and best shown in the sections stained by Marchi's method. In longitudinal and transverse sections under low power and high power was seen this somewhat peculiar appearance,—here and there little patches, consisting of rows of black dots, somewhat resembling patches of striation, in the muscle of the heart in fatty degeneration. The rows of dots were not in the muscle-columns, but in the spaces between them. In some the fat droplets had become very numerous, others represented earlier stages. In all the transverse striation could be seen, while the longitudinal striation simply looked a little coarse. He was not sure what relation this had to ordinary changes in muscle in ordinary parenchymatous neuritis, because it was a new reaction, and he could not find a description of the condition. He did not know whether it was peculiar to diabetes, or one likely to be found in muscular degeneration after parenchymatous neuritis.

*Dr James* said he had four cases of diabetes in his ward at once, of which one was a good example of neuritis with well-marked

drop-foot, but not much interference with sensation. He had been trying to concoct a theory of this, but could not very well do it. He had a case of advanced phthisis in his ward, undoubted tubercular lung disease, with exactly the same condition of drop-foot and also tenderness about the ankles, but not much sensory disturbance. Of course, neuritis had been described as occurring in tubercular disease, but he did not think it occurred nearly so frequently as in diabetes. As regards any toxic theory, at least with reference to the sugar, the occurrence of neuritis in tuberculosis put that out of account. Both the cases mentioned were recent.

*Dr Gibson* said that in listening to this admirable paper he was much impressed by the fact that the multiple neuritis occurring in diabetes seemed to be independent of the amount of sugar present. It appeared to be as marked in cases with small percentage as in those with large percentage. One could not help being struck with the analogy between what took place in alcoholic poisoning and what took place in this condition, and he thought most would agree that it must be some form of toxæmia that produced the neuritis. They knew well that in alcoholic poisoning there was often a mental affection of cortical centres. They had the same in diabetic coma. On the other hand, in alcoholic poisoning, but not quite so commonly, and more in the insidious forms, there was peripheral neuritis. The same might take place in this disease, and he thought there could be little doubt that from hyperacidity of the blood it was the presence of diacetic acid which might lead on through fermentative processes to presence of acetone. He thought one must agree with Prof. Fraser that some toxic agency was at the root of this change. He thought that observations of diabetes led to a practical warning. They could not fail to notice that a number of patients who were getting on pretty well before admission to hospital passed away rapidly after admission. Rigid dietary, he thought, was to blame for this. The rigid diet tended to produce more acidity, and in this and also in the cutting off the amount of fluid they had a conjunction of most favourable circumstances whereby the patient's blood might become a perfect laboratory for fermentative processes, of which acetone was the last development. He thought he was not going too far in making this statement. It was one of the matters that had attracted his attention, and therefore, like the pendulum, he had perhaps gone too much the other way with regard to the dietary in such cases.

*Dr Leith* said he had only examined one case of somewhat similar character, and had not yet completed the observations that he wished to make upon it. They confirmed those of Dr Bruce generally, but he could not say exactly to what extent.

*Dr Muir* said he would just like to join with the others in thanking Prof. Fraser. He thought it was important that such cases should be brought before them. Their knowledge of changes

in central nervous system, spinal cord, and nerves had been gradually widening of late, and it was of great importance to the scientific man and to the practitioner to have the knowledge that definite changes were liable to occur in the course of certain diseases. They had now quite a number of diseases in which there was parenchymatous change in the peripheral nerves. Perhaps the best example of that fully worked out in recent years was diphtheria. The direct proof by injection of chemical products, and the induction of parenchymatous degeneration in peripheral nerves in a very short time was, he thought, a very striking fact indeed, and one which brought into clear prominence facts which they knew before from a clinical point of view. Dr Bruce had described the appearances very fully and accurately. He (Dr Muir) had had an opportunity of examining the specimens before the meeting, and could fully concur with what he had said with regard to these changes. The changes in muscle, he thought, were of great interest, and it was rather striking that with the extensive changes in nerves and muscles the power of motion and sensation should not have been more impaired than they were in these cases.

*Dr J. Ritchie* said he quite agreed with what Dr Gibson had said, that it was a great mistake to limit the diet too rapidly, and also to limit the amount of fluid. The amount of sugar does not depend on the polyuria, but the sugar determines it. Dr Gibson had expressed the opinion that possibly the rigid diet was the cause of patients getting worse in the Infirmary. Frequently patients sought additional advice because they were becoming worse, therefore he did not think they were justified in drawing the conclusion that patients were getting worse because of the diet. It might be that they were getting worse before they sought admission to the Infirmary.

*Dr Church* said he had seen some cases in which diabetes had followed influenza. It had just occurred to him whether the microbe of influenza might not sometimes be the factor in the production of this nervous pathological condition. He referred more particularly to cases of pancreatic glycosuria, which seemed, in his experience, to be benefited by pancreatic extract freshly prepared. The result of treatment in Dr Fraser's case favoured pancreatic feeding.

*The President* said one could not help correlating these peripheral changes with certain cortical effects observed in diabetes. There was a very rare form of insanity called diabetic insanity, a melancholia usually. In one such case one of their staff, Dr Camshell Clark, found very decided changes in the nerve cells of the cortex. Had Dr Bruce found any such changes? A similar cause acting on the cortex might explain the mental changes in insanity. One might also fairly correlate with similar causes the occurrence of diabetic coma and congestive effects no doubt arising in the vaso-motor centres of the cortex, and, no doubt,

explicable on the toxic theory. A third series of phenomena were the boils and skin ulcerations. Was it not possible that in these cases also they had examples of toxic changes in the trophic nerves to the skin? One had seen the universal occurrence of boils and skin changes all over the body. He expressed entire concurrence with the other speakers in their opinion of the high value and interest of this paper.

*Prof. Fraser*, in reply, said that he did not know that he could add much to what had been said, but with regard to the point last referred to, viz., the possible occurrence of recognisable structural changes in the central nervous system in connexion with diabetes, no doubt the explanation which the President had given must be the correct one. All the phenomena of diabetes were, he thought, to be explained by some toxic substance. He was certain it was not acetone. It was a substance, however, which in the meantime had eluded observation. It was possibly the same product as was produced by alcohol in alcoholic neuritis. In the case of diabetes that product was derived from glucose, which first underwent alcoholic fermentation,—the patient, although a teetotaler, producing alcohol in his own body because of the glucose which was present. It acted as toxically on him as if he had imbibed it in the form of whisky or other alcoholic beverage. Dr James had referred to the difficulty of explaining the *possible*—he did not think that Dr James had gone any further—the possible neuritis that occurred in tubercular phthisis, on the ground that there could be no toxic substance; but that surely was not an obvious impossibility. There was undoubtedly a toxine in tubercle, produced by the tubercular process, and it was quite within the bounds of possibility that this toxine, as in the case of the toxine of diphtheria or the much less well-known toxine of influenza, might exert a degenerative influence on the nerve textures and produce neuritis in the same way. Peripheral neuritis was probably an affection associated with a great many more diseases than had hitherto been recognised. He was glad that the toxic theory had appeared to commend itself generally to those who had spoken. In the meantime, it seemed to be the only theory that coincided with the facts.



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L A W S

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF

E D I N B U R G H.

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INSTITUTED 1821.

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Printed for the Society

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1896.



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# L A W S.

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## I.—NAME AND OBJECTS OF THE SOCIETY.

1. The Society shall be denominated THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

2. The Objects of the Society shall be—To receive communications on Medicine and Surgery, and subjects connected therewith; to converse on medical topics; and to promote professional improvement by any other means that may, from time to time, be approved by the Society.

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## II.—CONSTITUTION OF THE SOCIETY.

The Society shall consist of Ordinary, Corresponding, and Honorary Members.

### I. OF ORDINARY MEMBERS.

1. Ordinary Members must be gentlemen legally qualified as medical practitioners.

2. Any person proposed as a Member must be recommended by two of the Ordinary Members of the Society. His name and qualifications must be intimated to the Senior Secretary at least fourteen days before the Meeting at which he is to be balloted for. Applications shall be intimated in the billet for the Ordinary Meeting thereafter, and then disposed of by ballot; and no person shall be declared elected unless three-fourths of the votes appear in his favour.

3. Every applicant for membership shall be required to lodge with the Treasurer the entry-money of One Guinea

previous to his name being proposed to the Society for admission, and this sum shall be retained by the Society in the event of his election.

4. The Ordinary Members, with the exception of those provided for in Law 5, shall pay an Annual Subscription of Ten Shillings ; they shall be entitled to take part in all the proceedings of the Society, and shall receive the billets calling the Meetings of the Society, and a copy of the Transactions free of charge.

5. The Ordinary Members residing beyond a radius of five miles from the General Post Office, Edinburgh, shall be exempted from paying the Annual Subscription, but shall not be entitled to take part in the private business of the Society, to hold office, or to receive the billets calling the Meetings, or a copy of the Transactions, *unless* they intimate to the Treasurer before the second Meeting of any Session their desire to participate in the full privileges of membership, and remit their Annual Subscription in due course.

6. The Ordinary Members exempted from paying the Annual Subscription under Law 5 may obtain the Transactions by an annual payment of Five Shillings, or such sum as the Council may from time to time determine.

7. Members who have intimated that they do not wish to avail themselves of the exemption provided for in Law 5 shall be liable for the Annual Subscription until they intimate to the contrary.

8. Members who are two years in arrears with the Annual Subscription shall receive intimation of the same from the Treasurer, and if payment be not made within one month, they shall, if within the five-mile radius, have their names removed from the list of Members of the Society, and if beyond that radius, they shall be considered as availing themselves of the provision in Law 5.

## 2. OF HONORARY MEMBERS.

1. The Honorary Members shall be gentlemen of distinguished attainments in Medicine or the allied sciences, who are not Ordinary Members of the Society. Their number shall be limited to Ten.

2. At the first Meeting in March of each Session, the vacancies in the list of Honorary Members shall be announced by the President, who shall on the same occasion invite the Members present to nominate fit and proper persons to supply the vacancies. The nomination list shall remain with the Secretaries for a fortnight thereafter for the admission of new names. The Council shall subsequently meet, and in the event of there being more than three nominations for each vacancy, the Council shall frame a list of three for each vacancy. This list shall be printed in the billets issued for the first Ordinary Meeting thereafter.

3. The voting upon the lists framed as above shall be taken at the first Ordinary Meeting thereafter, the lists of candidates nominated, as finally arranged, being announced by the President. Each Member present shall be entitled to vote for one name in the case of each vacancy; and the persons having the greatest number of votes so taken shall be declared to have the majority of votes of the Society, and to be appointed Honorary Members.

4. Every person elected an Honorary Member shall have immediate notice sent him by the Senior Secretary, along with a Diploma of the Society.

## 3. OF CORRESPONDING MEMBERS.

1. The Corresponding Members shall be gentlemen distinguished in Medical Science, who are not Ordinary Members of the Society.

2. At the first Meeting in March of each Session, any four Ordinary Members may propose in writing the name of any individual whom they deem suitable for the Diploma of Corresponding Member. The names proposed shall be submitted to the Council, and, if approved of, shall be printed in the billets for the first Ordinary Meeting thereafter, and be then balloted for. No person shall be declared elected unless two-thirds of the votes appear in his favour.

4. Every person elected a Corresponding Member shall have immediate notice sent him by the Senior Secretary, along with a Diploma of the Society.

#### 4. DIPLOMAS.

The Society shall grant to Honorary and Corresponding Members a Diploma to the following effect:—*"Societas Medico-Chirurgica Edinburgensis, anno salutis 1821 instituta, omnibus ad quos hæc pervenerint salutem. Eximium ornatissimumque virum. . . . quem scientiam medicam ingenio felici et laudabili diligentia coluisse compertum habemus, in numerum Sociorum (externorum, honorariorum) adscripsimus. In cujus rei fidem, has literas, manu præsidis nostri subscriptas, emitti lubenter jussimus.*

..... Præses.

..... }  
 ..... } a Secretis.

*"Edinburgi Anno Domini  
 Die."*

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### III.—OFFICE-BEARERS AND COUNCIL.

1. None but Ordinary Members entitled to vote can hold office in the Society.



2. The Office-bearers shall consist of a President, three Vice-Presidents, two Secretaries, a Treasurer, and an Editor of Transactions, who, together with eight other Members, shall constitute the Council.

3. The Election of the Office-bearers shall take place by ballot at the first Meeting in November.

4. Five Members of the Council shall constitute a quorum.

5. The Council shall regulate the business of the Society, Council. including the ordinary necessary expenditure. But it shall be in the power of six Ordinary Members, concurring, to bring any act of the Council under the review of the Society, provided they intimate their intention within one month from the date of the act.

6. Four Members of the Council, exclusive of the Office-bearers, shall go out of office annually, and shall not be again eligible for a year.

7. The President shall continue in office for two years. President.

8. One of the Vice-Presidents shall go out of office annually in rotation. Vice-Presidents.

9. The Secretaries, Treasurer, and Editor of Transactions shall be elected annually.

10. The duties of the Secretaries shall be as follows :— Secretaries.

The Senior Secretary shall have the management of the correspondence of the Society and of the Meetings of Council ; he shall receive all notices of Communications, of Patients, Pathological Specimens, etc., and shall arrange generally the public and private business of the Society's Meetings.

The Junior Secretary shall take Minutes which shall report the business, both public and private, of each Meeting of the Society.

- Treasurer. 11. (a) The Treasurer shall collect all subscriptions, income, and other moneys due to the Society, and shall make all payments on behalf of the Society.
- (b) The Funds of the Society shall be lodged with one of the Scotch Chartered Banks in Edinburgh in name of the Treasurer in his official capacity. He shall be entitled to draw upon the Bank Account, but that only for the purposes of the Society.
- (c) The Council may recommend that any part of the accumulated Funds be invested in any security recognised by the law of Scotland as suitable for Trustees.
- (d) After such recommendation has been approved of by the Society at one of its Ordinary Meetings, the Treasurer shall proceed to make such investments in the name of three Members of the Society who shall be nominated at an Ordinary Meeting.
- (e) These Members so nominated shall hold such investments as Trustees for behoof of the Society, and they and the survivors of them shall be bound, when called upon by a vote of the Society, to transfer the same to any other person or persons, or to realise the investments and pay the proceeds to the Treasurer.
- (f) The Treasurer and Council and the Members holding investments for behoof of the Society shall not be responsible collectively or individually for depreciation or loss in any investment made, held, or transferred by them in accordance with the foregoing rules, but each shall be held responsible for his own personal intrusions with the Funds of the Society.

12. A statement of the Funds shall be presented to the Society by the Treasurer at the Meeting in November or December of each year. At that Meeting the Society shall appoint a Committee to audit the Accounts ; and the Financial Statement, duly vouched, shall be printed and circulated among the Members along with the billet for the next Meeting of the Society.

13. The Editor of Transactions shall have charge of all matters connected with the obtaining, arranging, printing, and publishing the materials for the Society's Transactions, under the general supervision of the Council. <sup>Editor of Transactions</sup>

14. The Office-bearers, on going out of office, shall immediately be re-eligible, with the exception specified, Chap. III., § 6.

15. All motions relating to new laws, or to alterations on existing laws, shall be made in writing at an Ordinary Meeting, printed in the billets for, and considered at the following Meeting. The concurrence of two-thirds of the Meeting shall be required in order that any such motion may become law.

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#### IV.—MEETINGS OF THE SOCIETY.

1. Each Session of the Society shall commence on the first Wednesday of November, and shall continue for nine months ; the Meetings to be held on the first Wednesday of each month, at 8 P.M., except when otherwise ordered by the Council.

2. Additional Meetings may be held when the Council think it necessary.

3. At Ordinary Meetings five shall be a quorum ; but twelve shall be necessary for the election of Members.

4. Members shall, as far as possible, give due notice to the Senior Secretary, at least Ten days previous to the Meeting, of such Patients, Pathological Specimens, etc., as they may propose to exhibit, so that a list of these may be included in the billets.

5. Members shall not occupy more than five minutes in the exhibition of Patients and Specimens, nor more than thirty minutes in reading a Paper, except at the request of the President.

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#### V.—VISITORS.

1. Each Member shall have the privilege of introducing one Visitor at each Ordinary Meeting.

2. Visitors may take part in the Discussion if individually called upon by the President, and are to retire at the commencement of Private Business.

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